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AUGUST 1955

THURSTON SCOTT WELTON, M.D., EDITOR

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AUGUST 1955

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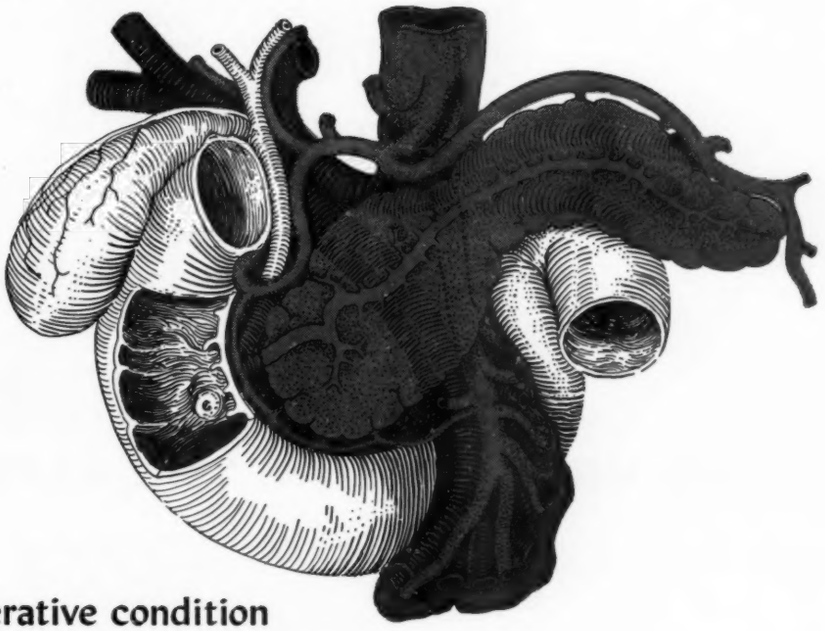

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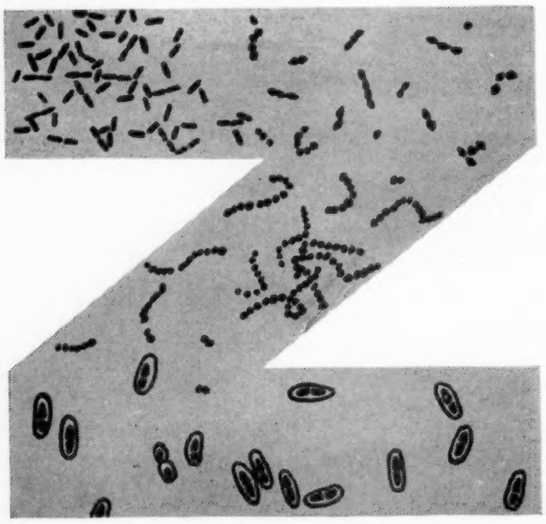

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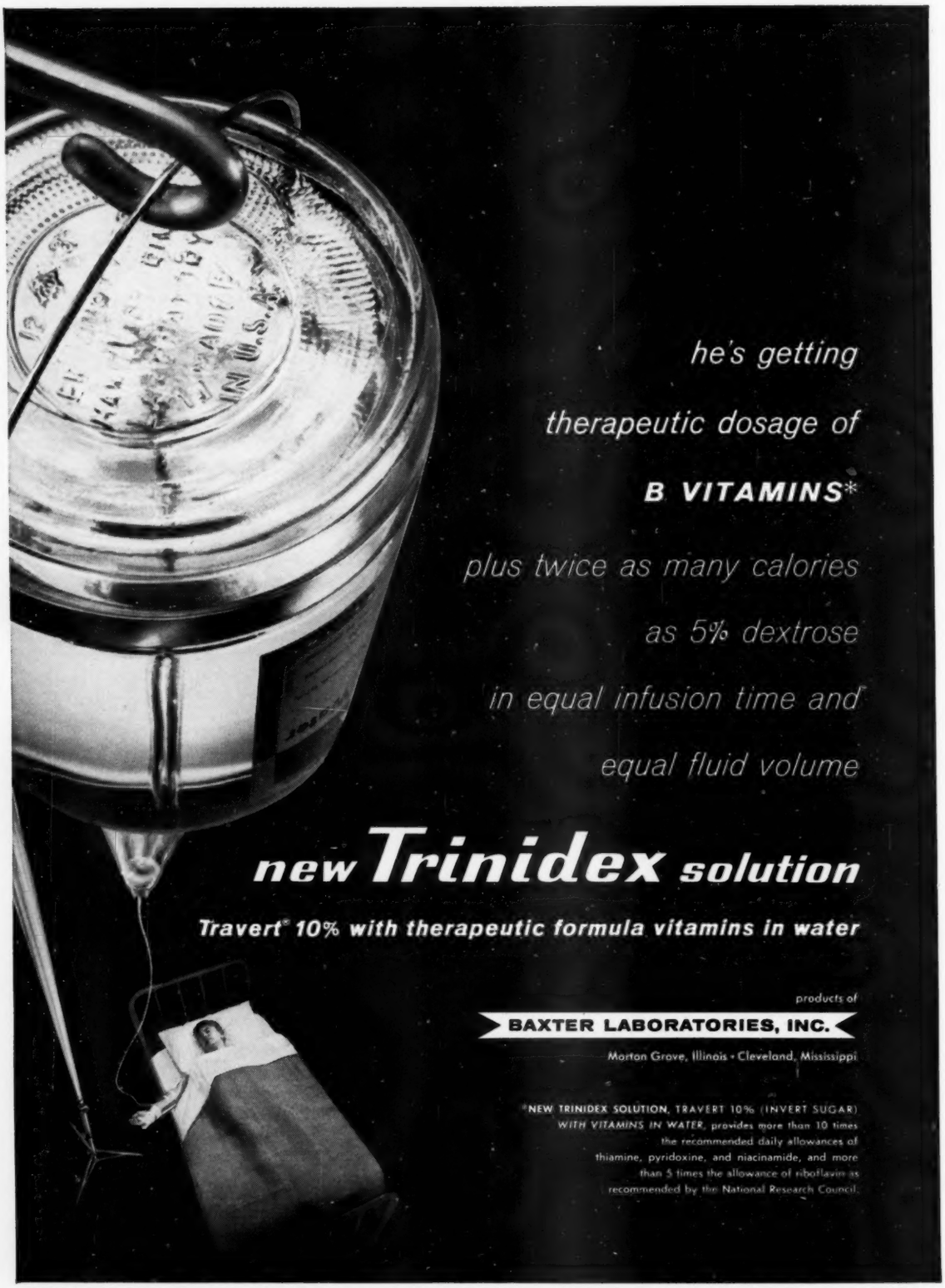
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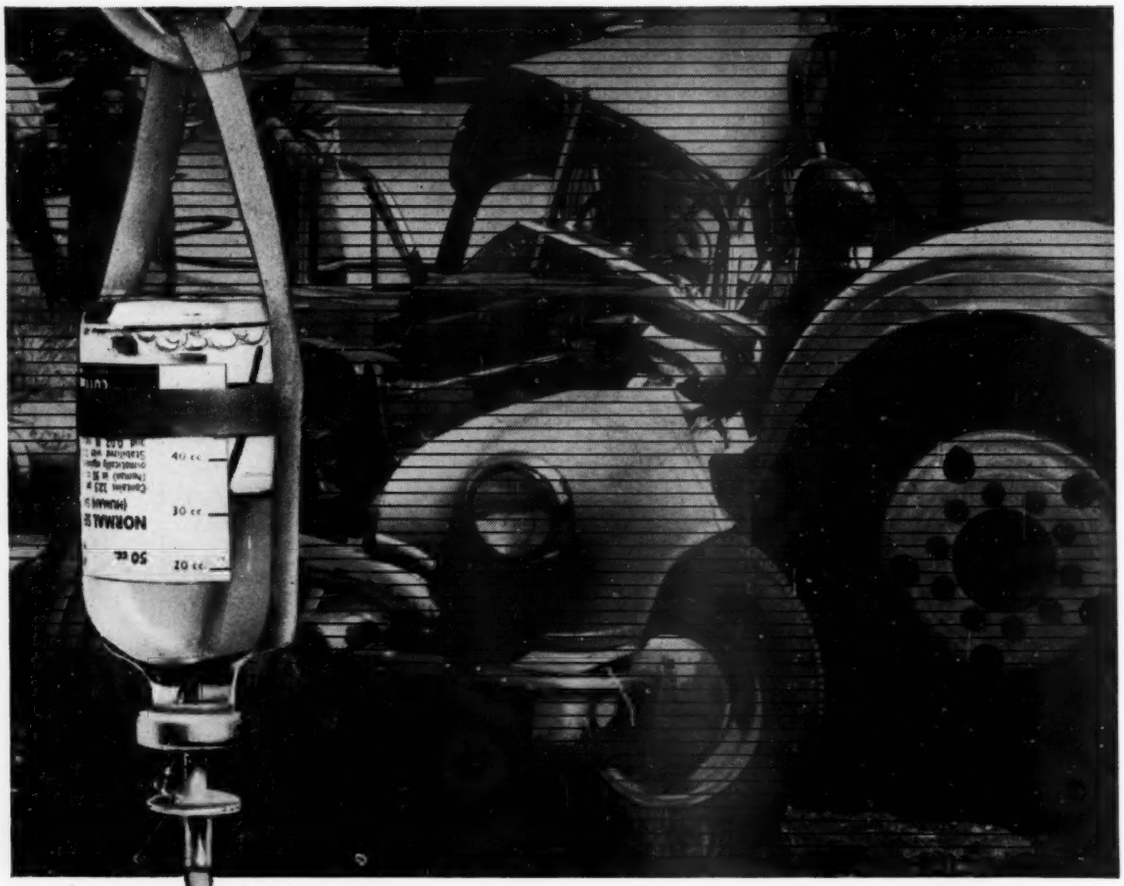

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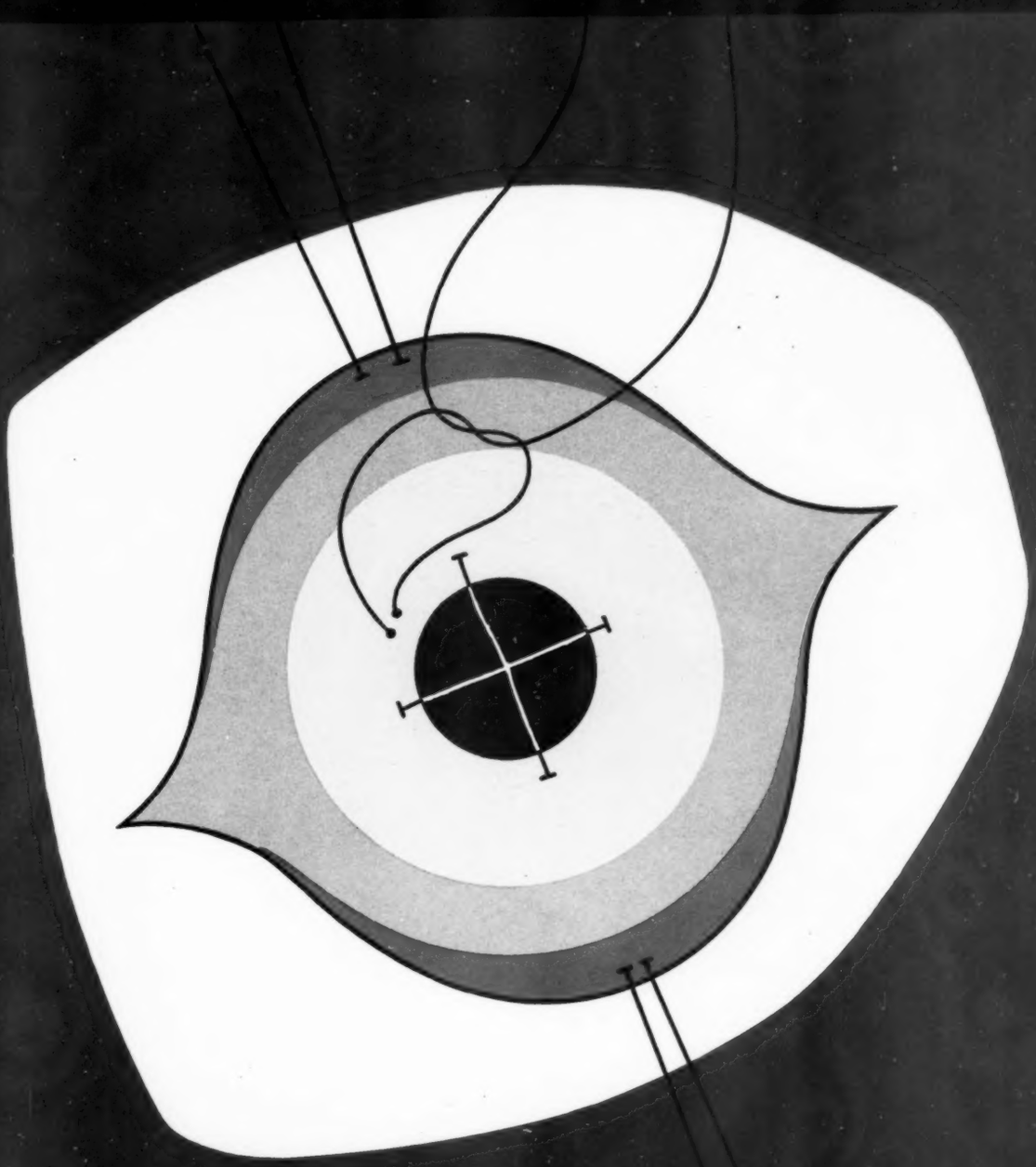

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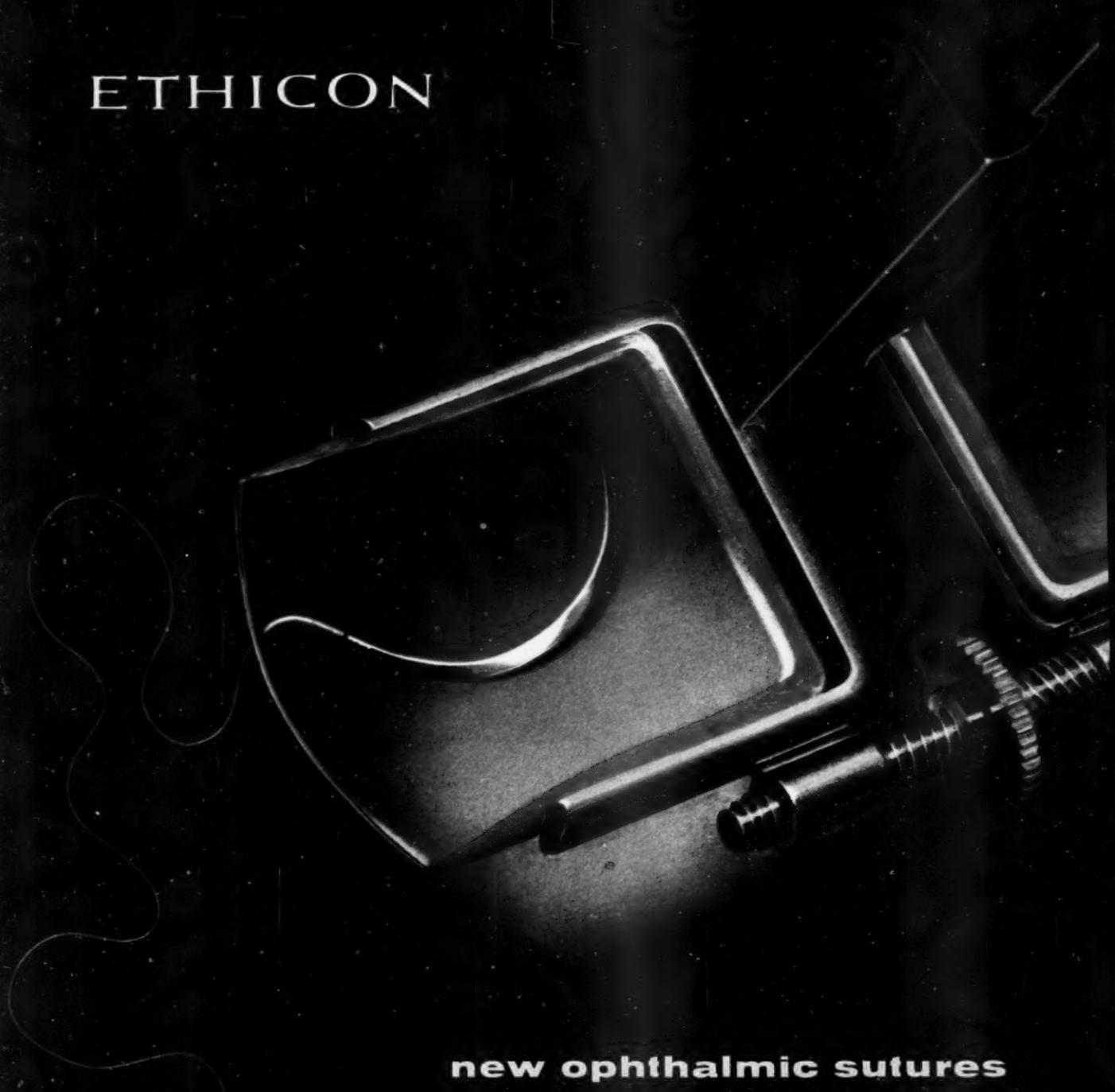

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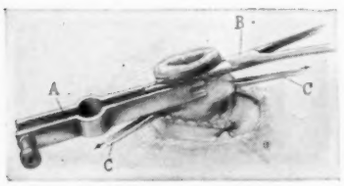

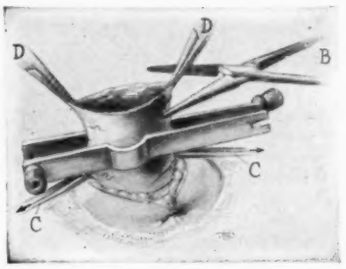

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Fig. 1—Wound closed and dressed around terminal bowel, forming colostomy. Daniel clamp (A) placed between large clamp (B) and two Allis clamps (C). *Fig. 2*—Daniel clamp (A) in place; bowel edges separated, held by Kelly forceps (D); original clamp (B) removed. *Fig. 3*—Size 28 or 30 soft-nose mushroom catheter (E) being inserted into bowel lumen. *Fig. 4*—Head of catheter is just below locked clamp. *Fig. 5*—Mushroom catheter advanced into abdominal portion of bowel under pressure of enema water.

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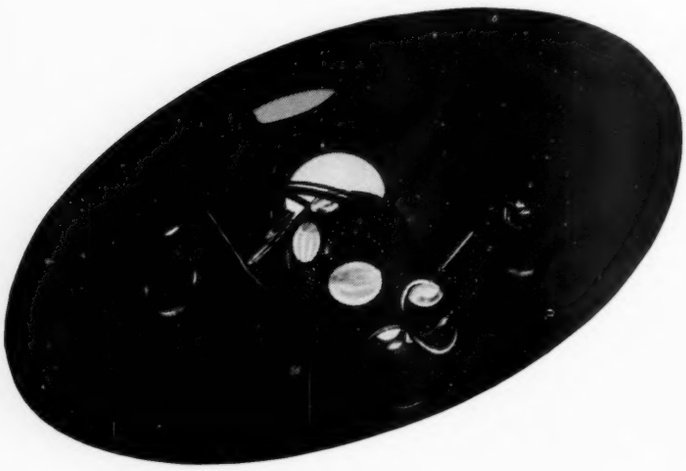

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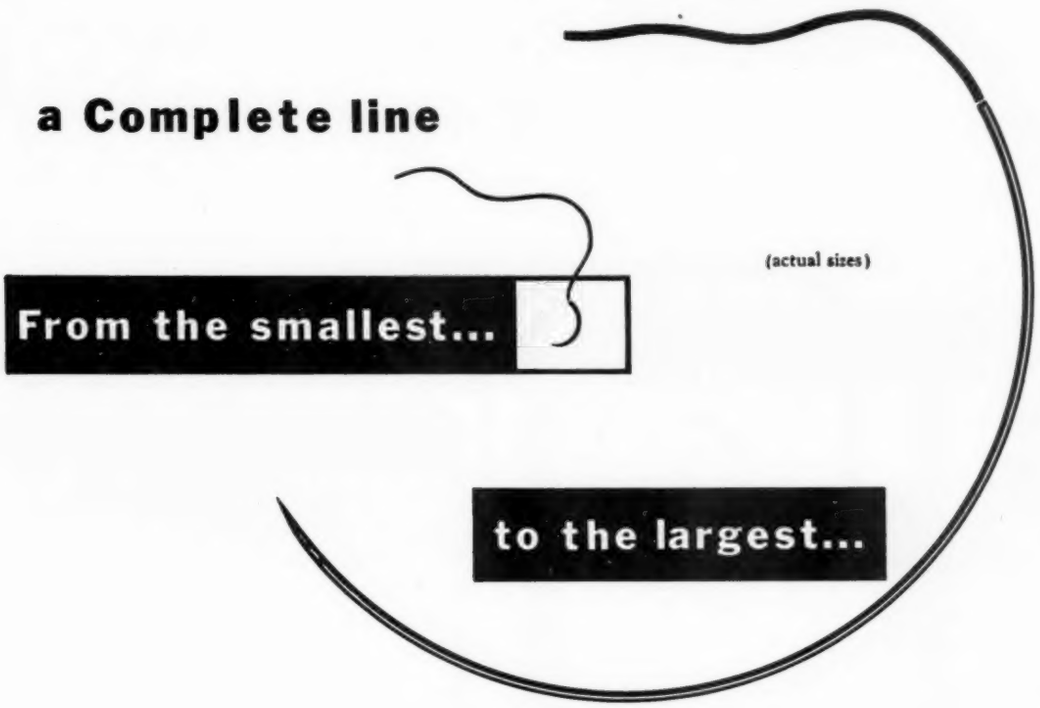

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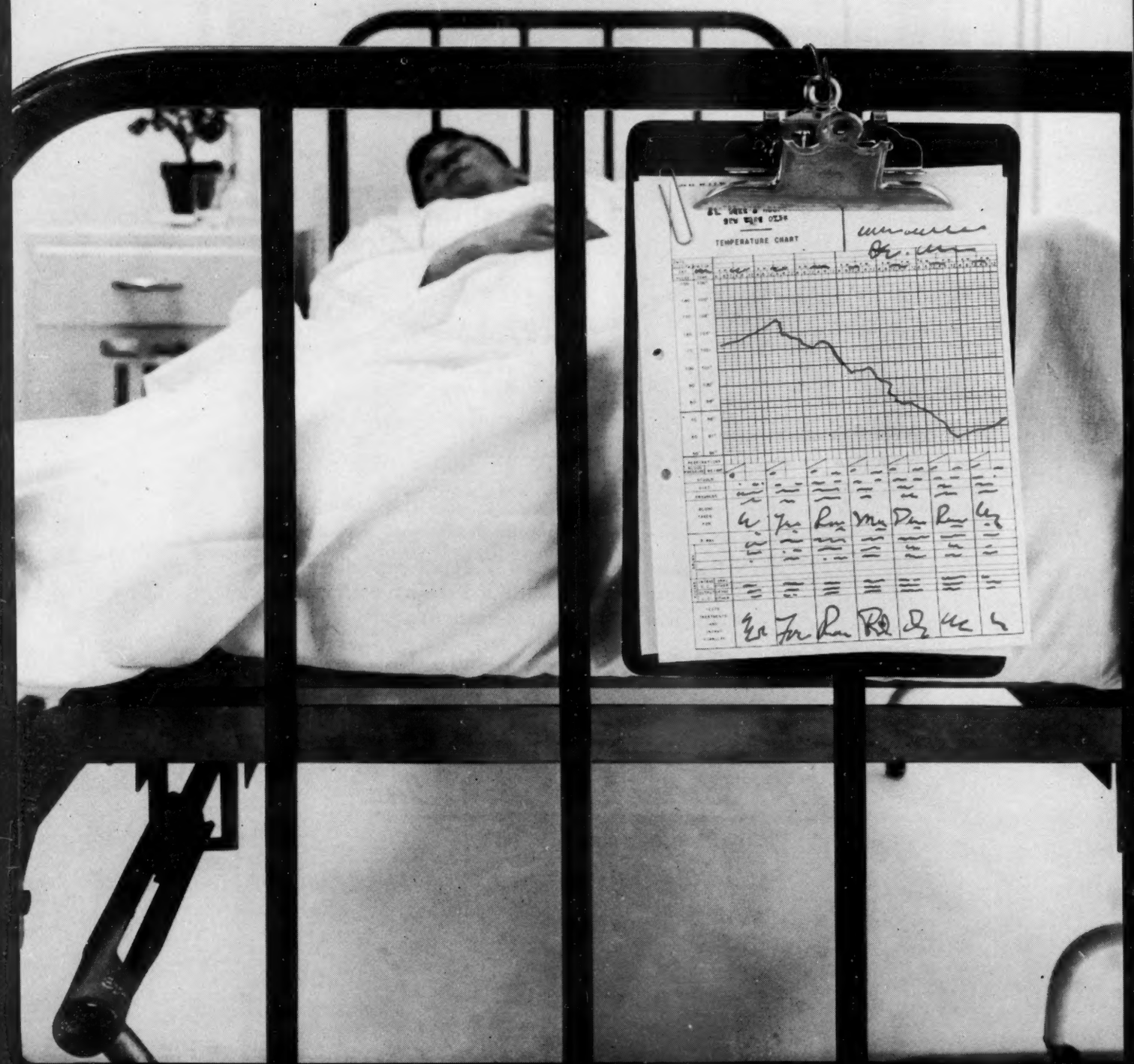

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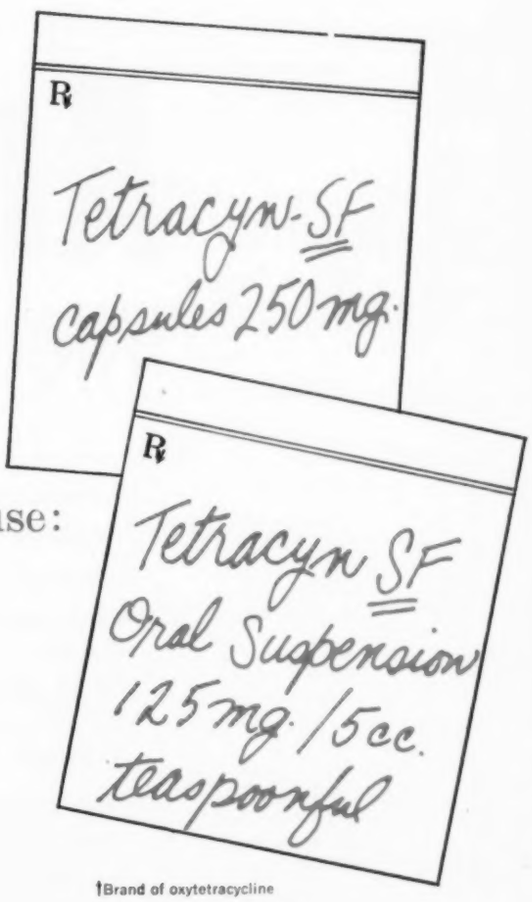

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1. Rechtman, A. M., and Yarrow, M. W.: Osteoporosis, Am. Pract. & Digest Treat. 5:691 (Sept.) 1954.
2. Cannon, P. R.; Frazier, L. E., and Hughes, R. H.: Factors Influencing Amino Acid Utilization in Tissue Protein Synthesis, in Symposium on Protein Metabolism, New York, The National Vitamin Foundation, Inc., 1954, pp. 55-90.

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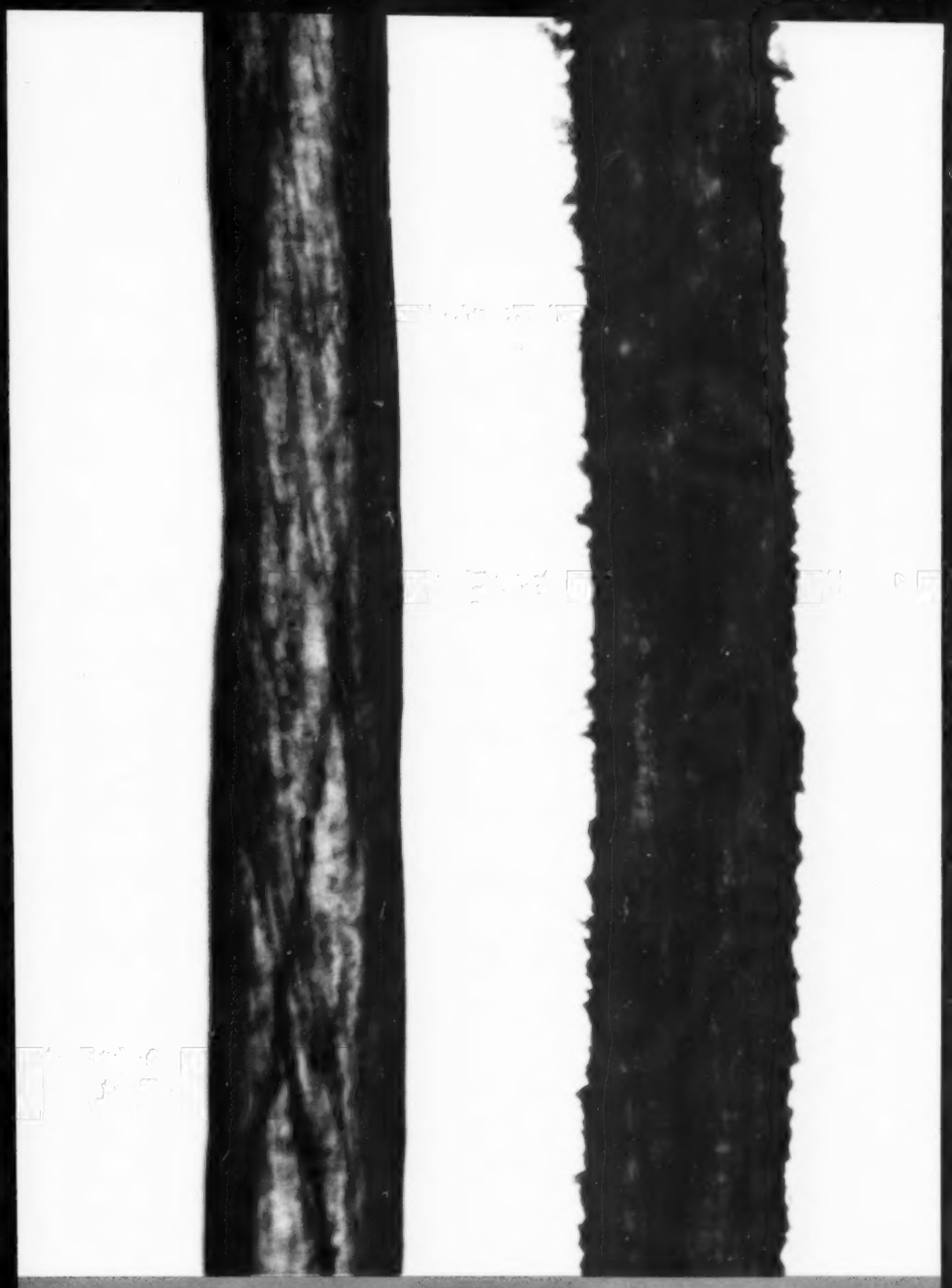

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Another leading gut

Photomicrograph reveals rough, frayed surface of another leading brand of gut. This has been ground to size. Gut processed in this way appears very uniform in diameter to the naked eye. But the photomicroscope reveals serious imperfections which may cause fraying and loss of strength when the knot is tied.

see exhibit on next page

Photomicrographs (unretouched) by E. J. Thomas, Stamford Laboratory of the Research Division of the American Cyanamid Company, Stamford, Conn.

Method used: dark field, transmitted bright field illumination, 138 x. Material used: medium chromic gut, size 5-0.

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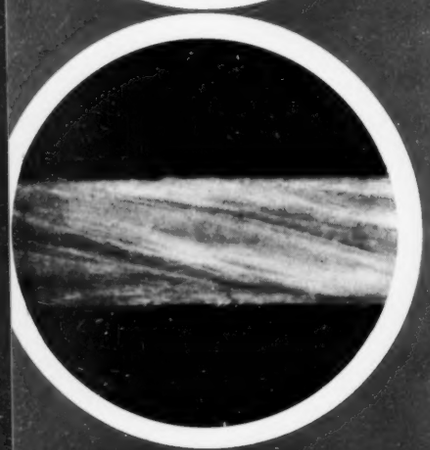

ANOTHER LEADING GUT

Photomicrograph detects separate and distinct plies in a strand of another leading brand of surgical gut. Here each ply was chromicized before they were twisted into suture strands. Such "ribbon chromicizing" hardens the surface of each ply, decreasing the natural bonding action, lowering the flexibility and tensile strength of the suture.

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Method used: dark field, reflected illumination, focus on crest of surface, 38 x. Material used: medium chromic gut, size 00.

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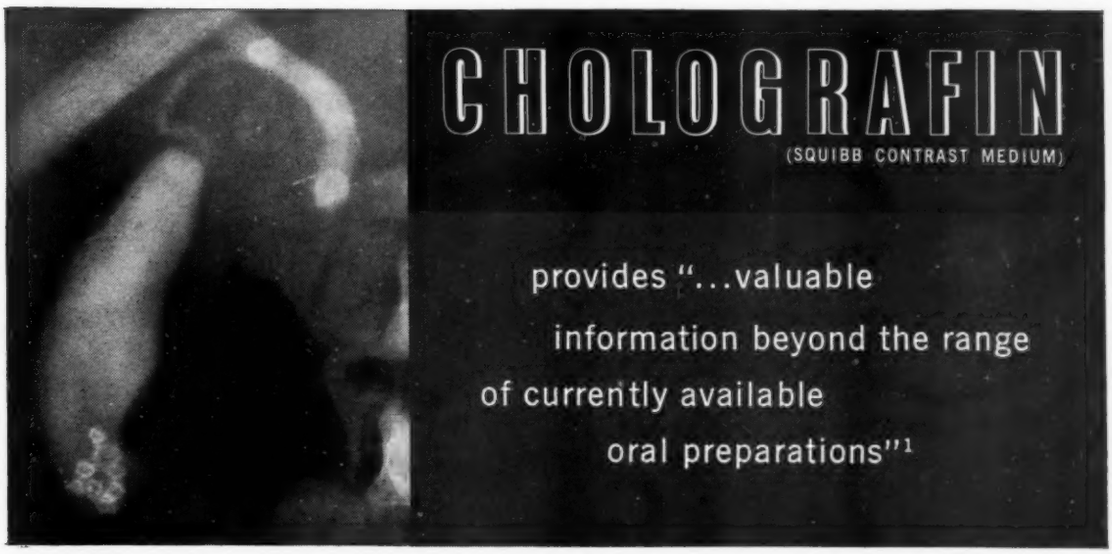

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1. Glenn, F., Evans, J., Hill, M., and McClenahan, J.: Ann. Surg. 140:600, 1954.

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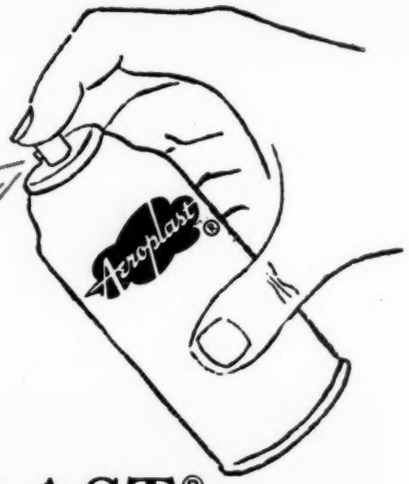

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1. Rigler, S. P. and Adams, W. E.: Experience with a new sprayable plastic as a dressing for operative wounds, Surg. 36:792 (Oct.) 1954. (University of Chicago Clinics, Chicago, Surgical Service).

2. Choy, D. S. J.: Clinical trials of a new plastic dressing for burns and surgical wounds, Arch. Surg. 68:33 (Jan.) 1954. (Bellevue Hospital, New York, Third Surgical Division—Dr. John Mulholland, chief).

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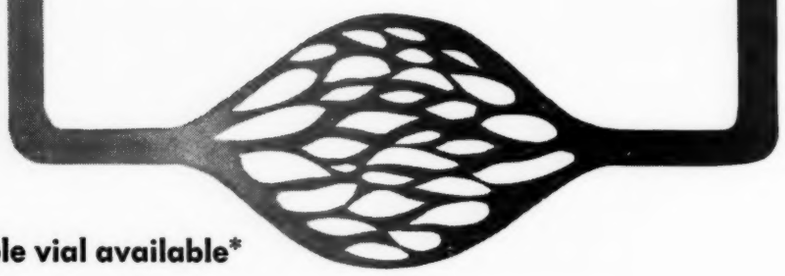

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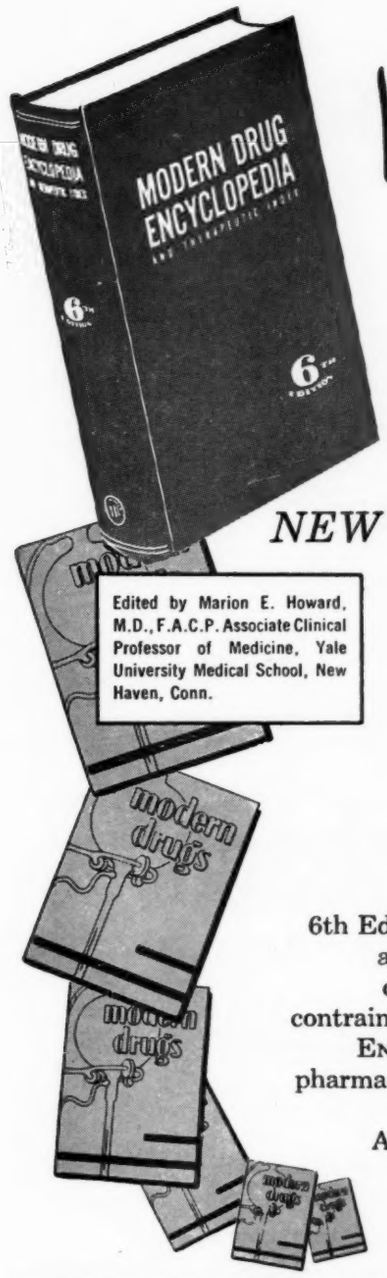

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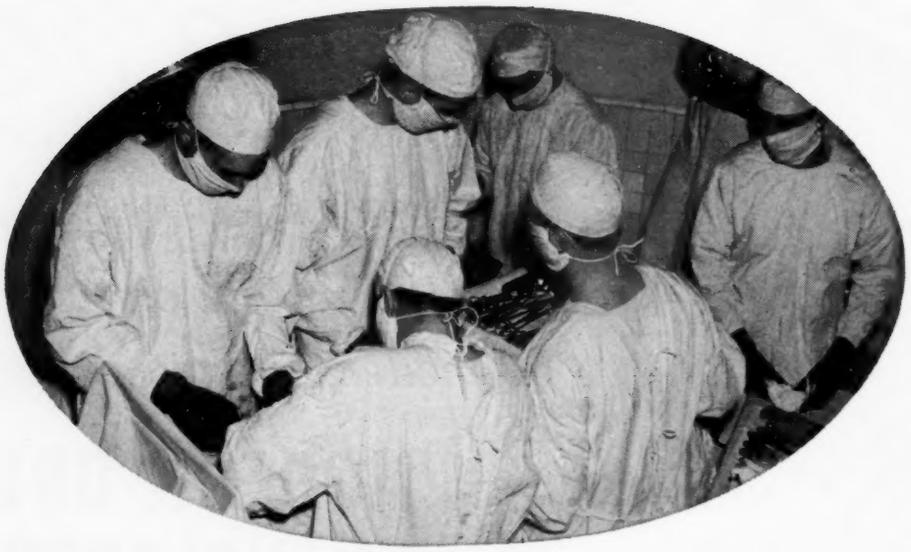

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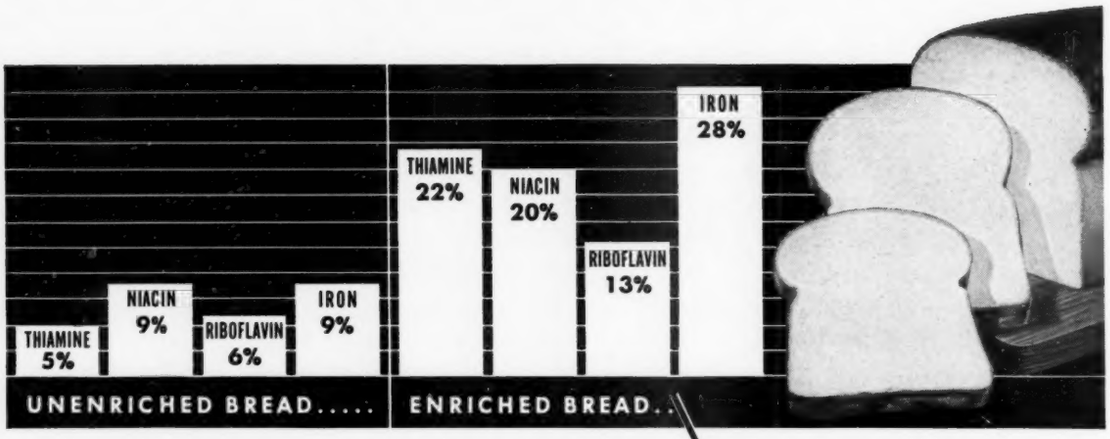

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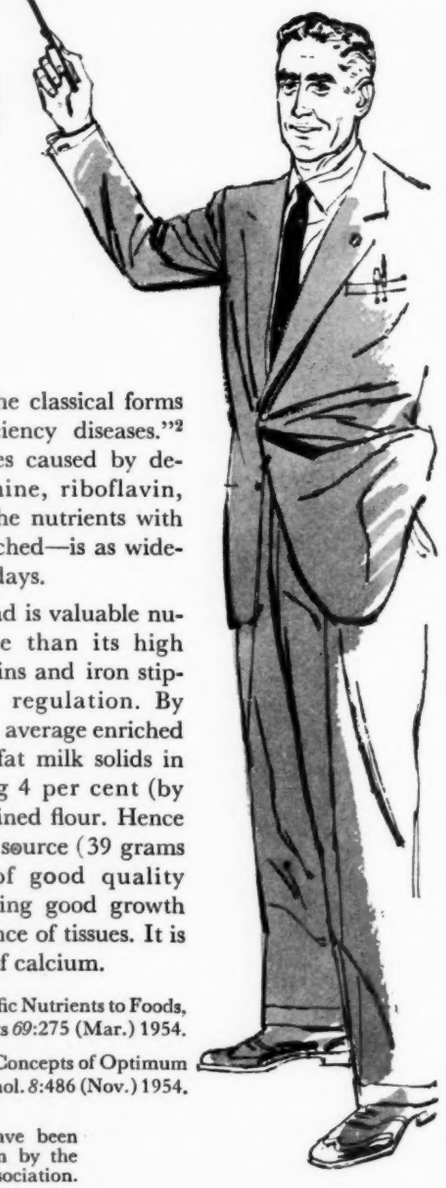

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1. The Addition of Specific Nutrients to Foods, Public Health Reports *69*:275 (Mar.) 1954.
2. King, C. G.: Newer Concepts of Optimum Nutrition, Food Technol. *8*:486 (Nov.) 1954.

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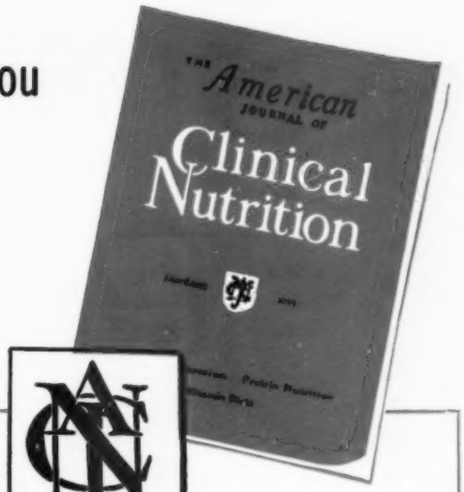

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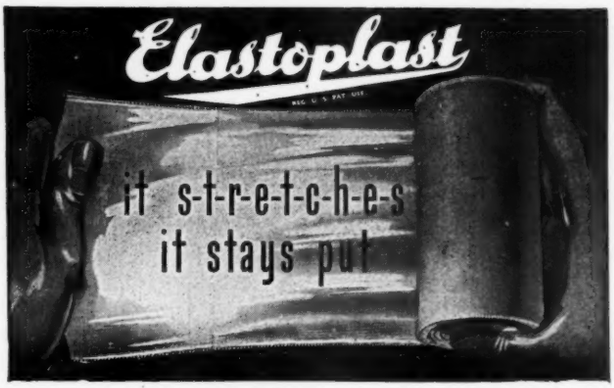

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Presidential Address

PROGRESS, PROBLEMS AND PROPOSALS

FIRST let me express my pride and happiness on being your President the past year. No greater honor has ever been given me and I am sincerely grateful for being placed among the great surgeons who have preceded me in this office. I am not unmindful of the splendid work done by the officers and members of committees of the Association, and give my thanks and appreciation to each of you.

The Pacific Coast Surgical Association was founded in San Francisco on April 11, 1925, almost thirty years ago. Anyone who reviews our activities, the scope of our discussions and the increasing eminence of our membership must be deeply impressed with the growth and advance of surgery during the life of our Association. I wish to present some facts and express some personal opinions about certain trends in medicine and the impact thereon of some of the changes in national affairs which have taken place during these thirty years. I use the term medicine in a broad sense to include medical education, research and practice.

There has been a staggering expansion of scientific knowledge, and factual information has accumulated at an amazing rate. Research in medicine has been more productive than at any other time in our history. Chemotherapy, antibiotics, substitution therapy in endocrine deficiencies, use of blood and its derivatives, new anesthetics, instruments and surgical technics are only a few of the triumphs during this period. Patients whose illnesses used to be considered hopeless and fatal are now treated with reasonable confidence of successful outcome in a large percentage of the population. Infant mortality has been reduced from 65 to 25 per 1,000 live births. Maternal mortality also has dropped from 7 to 1 per 1,000 deliveries. In 1953 there were 45,400,000 children under fifteen years of age. This is about 28 per cent of the population of our country. One federal bureau reports that more than 1,000,000 male workers will reach the age of sixty-five years during 1954–1955. The prolongation of life, the improved health of a large aging population, rehabilitation of the disabled of any age, the use of psychosomatic medicine in everyday practice and an increased application of preventive medicine in industry and the community have developed at an impressive rate during the past thirty years. This has been done by doctors of medicine who are far better trained and qualified as personal physicians or specialists than were their predecessors in the days of World War I.

All of this sounds wonderful and it is truly wonderful, for which all of us are grateful. However, during this period of improvement and expansion of medicine in the United States several conditions have developed which exert tremendous influences on us and our profession, and not all of these influences are good.

One of the greatest of these is the state of the Nation's economy; industrial productivity, employment, cost of living, wages and salaries, taxes and the ever changing purchasing power of the American dollar. We have experienced a financial boom, a national depression, artificial restoration by deficit financing, international

war and a postwar boom with increased production and continued inflation. Each of these changes has affected medicine and medical practice, and except for the brutal tragedies of war, it has been a thrilling period in which to live.

The depression emphasized an awareness of the unequal distribution of health facilities and medical care on the basis of cost. Many studies were made. Various programs of health service were initiated, including those paid for by taxes in whole or in part, and others on a voluntary basis varying from comprehensive medical care programs to prepaid hospitalization or insurance to cover part of the costs of illness.

Prepaid hospital insurance began in 1932. It has mushroomed into a national organization known as The Blue Cross. As the value of hospital insurance became recognized, medical care, on a prepaid voluntary basis, was added to the hospital insurance program. Labor organizations promptly recognized its value and included contributions by industry (the employer) to provide health care, at least in part, exclusive of industrial employment compensation problems, for their members. The United Mine Workers of America established one of the first of these programs. Organized labor in one industry after another obtained health benefits in addition to wages for their members. Government health services also expanded through the public health agencies, veterans administration and by various state laws which increased medical care for special diseases, rehabilitation of the handicapped, etc. Today at least 92,000,000 Americans have prepaid hospital and/or health insurance on a voluntary basis in one form or another. Much of this is paid for by the employer, included as a fringe benefit with wages at no financial cost to the recipient. A recent survey in California reports that 82 per cent of the members of unions in this state receive health and welfare benefits, and 51 per cent of them receive similar benefits for their dependents. These benefits are paid for entirely by their employers. The number of people encompassed by the several forms of voluntary prepaid hospital and/or health care increases year by year.

Our government provides fairly comprehensive health care from tax funds to about 21 million veterans, 5 million indigent, 3 million members of the armed forces and some 2 million dependents of military personnel. A total of 31 million are eligible to receive medical and hospital care from tax funds through various departments of the government. Thus about 123 million people have their health services provided in part, or a few of them in whole, by these two systems.

Another development in the field of prepaid hospital insurance in California is the Unemployment Compensation Disability Act enacted by the California Legislature in 1945. The last amendment to this Act became effective on January 1, 1954. This law requires all employees to pay 1 per cent of their wages to a fund which provides compensation for unemployment due to non-industrial illnesses and further provides the sum of $10. per day to the unemployed worker for a maximum of twelve days' hospitalization. This law combines hospital insurance with unemployment compensation and is in effect in three other states. Most students of tax-supported health insurance consider this trend very hazardous and one of the early steps up the ladder to complete compulsory health insurance, generally referred to as State Medicine.

World War II had a tremendous impact on medical education. From 1941 until the summer of 1946, our medical schools were in continuous session on what was called "the accelerated program." All male students in our medical schools, except a few physically unacceptable for military duty, soon were in uniform under the A.S.T.P. or NAVY V-12 programs. About 35 per cent of the faculty members were away from the school on active military duty during the period of the accelerated program. A medical student's schedule consisted of four nine-month sessions followed by an internship of nine months' duration. Under this system 31,651 students finished medical school. They, and about 4,000 members of the intern and resident staffs, provided more than 35,000 young physicians who entered the Armed Forces without previous experience in the private practice of medicine. Professional habits and attitudes developed during non-combat military experiences were well established when these young medical officers returned to civilian life. The system of regular hours of duty, relief by an officer of the day, periods of free time at night and weekends, was anticipated in civilian practice by many of them. A large percentage of the physicians who were graduated under this accelerated program sought graduate training in a medical specialty when they were

returned to civilian life. Some did this not to become experts in a special branch of medicine but primarily with the belief that certification by a specialty board would permit them to charge higher fees, enhance their chances of appointment to hospital staffs or, in general, raise the level of their standing in their profession.

War, also, called for intensive research, both fundamental and applied, in many branches of medicine. Investigations were pursued intensively and diligently. Many discoveries, vast amounts of new knowledge and rapid advances in medical care have resulted from these investigations. Wartime research, for the most part, was financed by federal funds in the form of grants-in-aid. This in itself had a vast influence on personnel because research budgets, prohibitive in size to a school, institution or an individual, were considered small expenditures at the federal budget level. This influence continued after the war was over and, in fact, the opportunity to provide funds for medical research increased amazingly. This in turn created an opportunity for some institutions and departments to include almost all kinds of expenses in applications for grants-in-aid if there was even a pretense at medical research. Some requests planned to transfer the costs of salaries, supplies, new equipment, etc., needed and desirable, from their regular school budgets to the short term financing by year-to-year grants-in-aid. Federal financing of research soon diverted funds from other sources previously available to medical investigators. Some individuals, foundations and industries discontinued or reduced their contributions for medical research, leaving investigators and their institutions dependent for the most part upon government aid for financing their medical research work.

The availability of government funds for medical research and the premium awarded in many academic circles to research accomplishments has a tendency to separate medical investigation from medical care and teaching. Many so-called investigators do little investigating but spend much of their time planning new projects for research assistants and budget requests for new grants-in-aid. Much of the real work is done by the members, junior grade, of the department or by technicians and technologists.

During the past thirty years undergraduate and graduate medical education has been influenced by national and medical economics, by the war and by military experiences. The impact of the accelerated program on medical schools and their graduates during World War II has been mentioned. Financing of medical schools and their hospitals and the scope of activities required of the faculties have changed remarkably during this time. Although medical schools do not have a uniform system of accounting, the cost per year of educating a medical student in 1925 was approximately $700. Today, in 1955, it is more than $3,000.

In addition, medical school faculties have been given responsibility for the education and training of a large part of the health personnel other than physicians. During the academic year 1953–1954 American medical schools listed 27,789 undergraduate medical students and 59,430 others as their student bodies. In so doing they lose a good deal of their effectiveness for instruction in a learned profession.

University administrations are pressing the medical schools to increase their enrollments, raise their tuition fees and to earn more and more of the budget moneys from the professional activities of members of the faculty. These activities overtax the faculties, divert their time and energy away from the teaching of students, interns and residents, and from research. A professor of surgery who is responsible for the daily care of from twenty to forty private patients in the hospital cannot do a very good job of training the next generation of physicians. Too much teaching at the medical school level is now done by last year's residents or inexperienced beginners on the faculty, not by the masters themselves.

Recent advances in medical knowledge, new technics and improved management of serious illnesses are based on science to a far greater degree than they were thirty years ago. The physician who makes a diagnosis "by ear" or begins his treatment by recollection of previous experience alone does not have a very good chance of anything better than average success. It may be true that the accumulation of health knowledge is too great for a single physician to master; therefore some specialization is inevitable and desirable. Specialization is unsound, however, when it becomes so narrow that it fragments the practice of medicine and the specialist loses an understanding and appreciation of the rest of medicine.

The American boards for medical specialties have influenced medicine in several ways during

this period. There are nineteen such boards issuing certificates in their own specialties and in at least fourteen subspecialties so we have specialties within a specialty. Organized with high ideals and lofty purposes, operated with enthusiasm and for the most part successfully, nevertheless the behavior of some of these boards has not been above reproach. All limit the field of professional activity of the diplomate. Some have attempted to limit the number of diplomates in their specialty. Most specialty boards give permanent approval once the candidate has passed his qualifying examinations. Most of them have required training programs by course content and length of time, including special "courses" in the so-called basic sciences, without reference to the availability of qualified teachers or facilities in these fields as applied to the ill patient. Hospitals frequently have been approved or disapproved for specialty training after inadequate surveys or on limited information about the qualifications of the teaching staff. Several government agencies, governing boards of many hospitals and some other organizations use certification by a specialty board as the main evaluation of competence and frequently give special opportunities, privileges and salaries to diplomates not available to competent physicians doing the same work but who do not possess a certificate from one of these boards.

The demand for specialty training increased tremendously after World War II although there has been a definite shift during the last two or three years toward more training for general practice. Existing residency programs were expanded; some increased the number of trainees but not the facilities, faculties or clinical material. Many hospitals which did not provide residency training before the war and which were unable to provide a satisfactory experience, entered the field for the first time. Among this latter group are many with a totally inadequate grasp of the basic meaning of graduate training. Most of these deficiencies in specialty training have been blamed on the hospitals and their staffs. This is not entirely true, however, as the members of the American specialty boards and to some degree the candidates or residents themselves have been partially responsible for the deficiencies in specialty training.

Another change in American life having a forceful impact on medicine is the migration of our population. This is true, especially in the Pacific states and particularly in California. During the dustbowl days of the middle thirties, thousands of families came West. Most of them were without sufficient funds to prevent them from becoming "medical indigents" in the event of serious illness to anyone in their family. Medical care for these people became a financial burden to the communities in California which were not legally responsible in any way for their presence or their medical attention. In April, 1938, a great experiment in the practice of medicine was launched in California by the creation of Agricultural Workers' Health and Medical Association, formed jointly by the California Medical Association and the Federal Farm Security Administration. The federal government made all of the social service and eligibility studies based on policies and regulations established by California physicians and lay members of the Board of Directors of the Association. California doctors and hospitals provided all of the care to these individuals. Our government paid all the bills with money appropriated from federal tax funds. Medical care was of a very high caliber. Abuses by patients or physicians was at a minimum. Operating costs were reasonable. Practically everyone connected with this service was well satisfied. The need for this organization disappeared with the return of financially good times and the agency was disbanded in 1940. By this time most of the new families had gone home or had established themselves permanently on the West Coast.

With the approach of World War II there was another shift of population to the West. Hundreds of thousands of workers came or were brought here for employment in war industries. This created a multitude of health problems in various areas, which were solved poorly on a temporary basis or not at all. Fortunately for us in California, most of these people returned home when the war was over.

In 1946 the greatest peacetime migration in the history of this country began with the arrival of thousands of new families seeking permanent residence and employment on the West Coast. By 1953 the population in California had increased more than 55 per cent in seven years. These newcomers sought permanent residence and jobs. They needed doctors and hospitals as well as homes and schools. During the year 1953–1954 more than 5,200 new school rooms, 100 every Monday morning, were built and opened in California.

It is a fact that good medicine has moved to the country in California and in many Western states. There are good hospitals and excellent physicians in many small cities. Physicians well trained for family practice, specialists in general surgery, pediatrics, obstetrics, orthopedics, neurosurgery, chest surgery and, in fact, almost all of the medical specialties are practicing in these communities. Many of them have built new hospitals or remodeled and expanded old ones. A very small part of our population lives more than about three hours' travel time by automobile from such health facilities. No longer are patients referred from these areas to big cities, large clinics or medical institutions except for very unusual conditions. Adequate care by experts is usually available near home.

Most of the influences I have mentioned are here to stay, in one form or another. National economics will continue to change and to influence medical education, research and practice as it changes. Prepaid medical and hospital care will not be dropped. Required military service, at least for short periods, seems inevitable for the foreseeable future. Medical schools will not be abandoned and the specialty boards seem well established. The American people will continue to move around and there is no end in sight for the migration of population to the Pacific Coast. Probably nothing in this world is wholly good or wholly bad, so out of these changes much good has come. What can be done to preserve and improve it? Revision of some opinions formed earlier in this period of economic changes may be necessary.

Superior medical education depends upon what I call the three M's: Men, Material with which to work and Money enough to make work possible. The greatest of these is Men. It is quality, not quantity, of personnel that counts. Medical students must be better grounded in the natural sciences than they were even fifteen years ago, otherwise they will be unable to cope with the rapidly advancing, usable knowledge which is making medicine more nearly an exact science each year. Medical school faculties should be composed of capable, interested teachers who are experts in their fields, sympathetic and enthusiastic for their work. Students should have close daily association with these teachers, most of whom should be of high academic rank rather than beginners. When Ex-President Herbert Hoover said, "The imperative need of this nation at all times is the leadership of the uncommon men or women," he had in mind, I am sure, the uncommon doctor as well as the politician, soldier or industrialist.

The educational costs of a medical school should be borne by the university, but I am convinced that the cost of operating hospitals and rendering medical care required to train the next generation of physicians is not a fair charge against the educational budget. Medical school hospitals and clinics should be supported by the medical profession at least by referring enough patients with various illnesses and diseases to satisfy the needs for teaching purposes. These patients, or some form of prepaid hospital insurance, should pay the hospital costs. Members of the faculty should be paid by the medical school as teachers and investigators but should not be expected to produce an income for themselves or the university from fees which might be collected for medical care rendered by student trainees under faculty direction. This principle has been used successfully by dentists and schools of dentistry in their clinics for half a century. Our medical schools would be benefited immeasurably by the adoption of a similar plan.

Medical research should be placed in its proper perspective and not be the major goal of the faculty nor receive the largest budget from the administration. Year-to-year grants-in-aid for research projects tend toward isolation into special fields of research with considerable separation from the activities of the school as a whole. Under this system medical schools have little chance to select and develop teams of research personnel for sustained, long term efforts in related fields of inquiry. What is needed is a reasonable amount of money regularly available for research and under the control of the medical school itself, so that anyone on the faculty who is interested and capable may have the time, the opportunity and a place to do his investigating.

Prepaid medical care, if it is going to remain on a voluntary basis and not lead us step by step into socialized medicine under some central, bureaucratic control, must fulfill three basic requirements:

1. The subscriber to health insurance must not be required to receive his medical and hospital care from physicians employed or facilities owned and operated by the agency selling the insurance. This holds true whether the agency

is a commercial company, a group of physicians, a university, or our government.

2. Some form of co-insurance must be required of the subscriber. This may be the payment of part or all of the premium, making the first visit to the physician deductible, or any of the several methods of co-insurance.

3. Medical and hospital insurance should be kept rigidly separate from compulsory unemployment compensation disability.

The so-called fringe benefits, which provide partial medical care and hospitalization paid for entirely by the employer, have at least one grave danger. What will happen in the event of serious depression and lengthy unemployment for any considerable number of our employable population? If such an economic disaster occurs, it seems only reasonable to expect that our population in the low income group will request and get some form of tax-supported and politically controlled medical care. The medical profession should take the leadership now in strengthening and expanding prepaid medical care and hospitalization on a voluntary basis. The three essentials mentioned heretofore and the principles and experiences of the Agricultural Workers' Health and Medical Association have proved themselves sound in meeting that problem.

Health services to veterans should be limited to care of service-connected illnesses except for the nonservice-connected illnesses for which the veteran is unable to pay.

The training of medical specialists can and should be improved. The temporary postwar excess of applicants and shortage of adequate facilities and qualified teachers no longer exist. Too many centers, given approval for residency training after the war, are unsatisfactory, and their graduates are readily identified as inadequate by the examiners of the specialty boards. Better certification and approval of training centers must be provided, eliminating those that under the pretense of specialty training are accepting young physicians for hospital service to the private patients of a group of busy practitioners. There must be a return to the residency training program which supplies adequate experience and graded responsibility under supervision of expert clinicians who are competent teachers. In this way the trainee will learn by doing and thereby develop maturity of judgment, technical competence, a critical attitude and confidence in his own ability.

I believe that education of the medical student, intern and resident is the responsibility of the faculty of a medical school. It is high time that facilities and personnel responsible for teaching interns and residents be identified and affiliated with our medical schools. Many hospitals, including some so-called "Deans' Committee" hospitals of the Veterans Administration, approved for independent residency programs in surgery, do not have the clinical material necessary for the complete training of a surgeon. They need the close affiliation with a medical school to meet these deficiencies of training. Rotation of residents through both services would provide adequate clinical material and fine teachers. Now that we have the Conference Committee on Graduate Training in Surgery, improvement in our training of surgeons should be less difficult. Regional committees whose members are thoroughly familiar with the hospitals and physicians in the local communities could be highly efficient in eliminating unqualified centers and recommending new ones able to do a superior job. Regional survey committees for internships, established by the Association of American Medical Colleges fifteen years ago, have proved eminently successful.

In the event of another war we should avoid the so-called accelerated program of teaching in our medical schools. Although this method delivered to the Armed Forces large numbers of young men with an M.D. degree, it was thoroughly unsound medical education. Teams of investigators, faculties and similar medical groups should be strengthened, not broken up or disbanded. It is reasonable to expect that if war comes again, military and civilian medicine will be coordinated into an over-all medical service for the emergency.

There is no doubt that the next thirty years will bring even more changes and greater impacts on medicine than have occurred during the life of this Society. There are many uncertainties at present but I have great confidence in the future—confidence in our ability to maintain leadership and increase our competence; to foresee and meet economic, industrial, scientific, educational, social, political and other changes with decision and action, blending them into a system of medicine that will remain the finest anywhere. The next thirty years, indeed, will be a thrilling period in which to live.

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Isolated Lipodystrophy, a Form of Mesenteric Tumor

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THE surgical extirpation of a mesenteric tumor can be a difficult and hazardous procedure. Proper management of such a lesion depends upon recognition of the differences between true neoplasms, both benign and malignant, and certain non-neoplastic tumorous processes of the mesentery. These latter conditions include Whipple's disease (intestinal lipodystrophy), the systemic variant of Weber-Christian disease (systemic nodular panniculitis), sclerosing lipogranuloma, tuberculous mesenteric lymphadenitis, and traumatic and pancreatic fat necrosis.

We have recently observed five cases of mesenteric tumor which differed sufficiently from the foregoing to suggest a unique entity.

CASE REPORTS

CASE I. E. B., a fifty-five year old woman, had experienced mild intermittent back pain for eight years. Two years prior to hospitalization the pain became more severe and shifted to the lower back. Because an abdominal mass was present on physical examination, the patient's local physician performed an exploratory laparotomy in August, 1953. A large tumorous process involving the small bowel mesentery was found and biopsied. Histologic interpretation was inconclusive; neoplasm could not be excluded. The patient was referred to the University of California Hospital for further consultation and treatment.

On admission the patient had no complaints referable to the gastrointestinal tract. Routine laboratory studies were within normal limits. Physical examination revealed the presence in the mid-abdomen of a large, firm mass which was relatively immobile but was thrust forward visibly by pulsations of the abdominal aorta. No bruit was heard. At operation the mesentery of the small intestine was found to be involved diffusely in a large tumorous mass which showed areas of nodular induration. The liver, pancreas and omentum were normal. The mass was considered to be neoplastic and an attempt was made to resect it. Although much of the small intestine and the right colon were sacrificed, the tumor could not be excised completely.

When last seen eighteen months after operation the patient had made a successful adjustment to her nutritional problem.

CASE II. J. H., a seventy-one year old man, was admitted to the University of California Hospital in August, 1954, with a history of epigastric pain for one month. Following the initial episode of pain several tarry stools were noted on subsequent days. The patient lost 12 pounds in one month and had considerable anorexia and vomiting the week prior to entry. One episode of hematemesis occurred shortly before admission.

X-ray study of the upper gastrointestinal tract demonstrated a filling defect in the antrum without evidence of obstruction. Abdominal examination showed an ill defined mass in the right epigastrium. At operation a large ulcer (5.0 cm. in diameter) was found on the lesser curvature of the stomach. The process had invaded through the gastric wall and involved the serosal surface. Lymph nodes were prominent along both curvatures of the stomach, in the region of the head of the pancreas, and about the celiac vessels. The spleen was enlarged and covered with a granu-

lar exudate. The liver and pancreas were normal. The root of the mesentery of the entire small intestine was nodular, indurated and thickened up to 9 cm. The process was diffuse, extending to within a few centimeters of the mesenteric border of the intestine, and had a distinctly yellow color with numerous bright yellow nodular and plaque-like lesions scattered throughout. There was no obvious distention of lymphatic pathways in the mesentery and no visible mesenteric lymphadenopathy. There was no evidence of a similar process in the omental or perivisceral fat, and the small and large intestines appeared grossly normal. A biopsy of this mesenteric lesion was made. The gastric lesion was removed by subtotal gastrectomy with restoration of intestinal continuity by gastroduodenostomy. The pathologic diagnosis was lymphosarcoma of the stomach.

During the six months since operation the patient completed a course of x-ray therapy to the upper abdomen and is now without symptoms referable to the primary disease.

Case III. E. H., a forty-eight year old woman, was admitted to a hospital in Tennessee with the chief complaints of polyuria and dysuria. Preliminary examination disclosed a palpable, ill defined abdominal mass. There was no abdominal tenderness or gastrointestinal symptoms. Other symptoms were minimal and did not appear to be related to the mass. Laboratory findings, including pancreatic function studies, were within normal range. An intravenous pyelogram was normal. The preoperative diagnosis was retroperitoneal tumor and the patient underwent exploratory laparotomy. At operation the mesentery appeared markedly thickened and nodular. It had an opaque, mottled, yellowish grey to bright orange color and was rubbery and firm. The adjacent small bowel was entirely normal and there was no evidence of lymph stasis or mesenteric lymphadenopathy. The liver and pancreas were grossly normal. A biopsy of the mesenteric mass was made.

During the year since operation the patient has lived in California. She has been well although the abdominal mass remains palpable.

Case IV. P. B., a sixty year old man, entered the hospital on October 9, 1936, with an infection of the hand which cleared promptly after incision and drainage. He remained well until January 22, 1941, when he was again admitted, this time with acute mastoiditis which responded well to surgical treatment. In January, 1942, the patient began to have back pain, weight loss, nasal obstruction and bleeding, attacks of angina, and ulcer symptoms, all of which proved to be manifestations of his primary disease, systemic amyloidosis. A large lesser curvature ulcer was subsequently demonstrated by roentgen examination and was treated successfully by dietary regimen. During the patient's last hospital stay in August, 1942, he died from progressive cardiovascular decompensation.

Autopsy revealed myocardial infarction secondary to coronary atherosclerosis. Extensive deposits of amyloid were found over the entire endocardium and cardiac valves. There was widespread deposition of this material in the lymph nodes, adrenals, liver, spleen, pancreas, skin, nasal mucosa, pulmonary artery and its branches, the aorta and many of the visceral arteries and arterioles. In the stomach was a large lesser curvature antral ulcer which was infiltrated extensively with amyloid. On the basis of these findings a diagnosis of primary systemic amyloidosis was made. The small and large intestines were normal. The liver showed focal central necrosis. The mesentery of the small bowel was thickened, nodular and firm. The fatty tissues throughout the rest of the body failed to show a similar change.

Case V. G. K., a fifty-two year old man, was admitted to the hospital in August, 1944, with the chief complaint of shortness of breath. The physical findings were an enlarged heart, slightly elevated blood pressure, basal rales, abdominal distention and dependent edema. X-rays showed cardiac enlargement and fluid in both pleural bases. Two weeks after admission a hard, nodular mass was felt in the right upper abdomen. A gastrointestinal series at that time revealed that the mass lay to the right of the duodenal bulb. A laparotomy was planned but postponed when the patient went into cardiac failure. Nineteen hundred centimeters of straw-colored fluid were removed from the abdomen, and the patient was treated for cardiac decompensation. The right upper quadrant mass remained palpable during the ensuing months and repeat gastrointestinal studies indicated that the mass was extrinsic to the gastrointestinal tract. The patient had had no previous gastrointestinal complaints but started to vomit shortly before death in November, 1944.

At autopsy fluid was present in the abdominal and right pleural cavities. The heart showed hypertrophy and dilatation, chronic myocardial and endocardial fibrosis, and mural thrombi in the left ventricle with an organizing thrombus in the pulmonary artery. Pulmonary congestion and edema with focal bronchopneumonia were observed, and the liver and spleen were congested. Chronic cholecystitis with cholelithiasis was also present. The small and large intestines were normal. However, the entire small bowel mesentery was greatly enlarged and contained large zones of opaque, yellowish fat necrosis. This rubbery, firm, greyish yellow fatty mass was rounded, about 8 cm. in thickness, and extended from the root of the mesentery to within a short distance of the mesenteric border of the small bowel. There was no evidence of alteration of fatty tissue elsewhere in the body.

COMMENTS

These five adult patients had no symptoms attributable to their abdominal masses, with the possible exception of the subjective complaints of backache in Cases I and IV. The symptoms of ulcer and weight loss present in Cases II and IV were explained by the subsequent discovery of a gastric ulcer at operation and autopsy, respectively. The gastric ulcers did not appear related in any way to the mesenteric lesion. Diarrhea was never a symptom in any case, and fever, abdominal pain and bowel obstruction all were notably absent. An abdominal mass was palpated in four cases, in three of which exploratory laparotomy was performed. The fourth patient died of cardiovascular disease before operation could be undertaken, and verification of the presence of the tumor in this case was made at postmortem examination. In Case V the mesenteric mass was an incidental autopsy finding, systemic amyloidosis constituting the major pathologic condition. In Case II the diagnosis of lymphosarcoma with secondary gastric ulceration was made at operation, at which time the mesenteric location of the palpable abdominal mass was revealed and the tissue biopsied for further study.

In all five cases the mesentery was involved in a massive tumorous process which extended from the root of the mesentery to within a few centimeters of the intestine proper. (Figs. 1 and 2.) This nodular fusiform mass attained a thickness of as much as 9 cm. at the root of the mesentery and thinned out somewhat distally. The zone immediately adjacent to the intestine was uniformly unaltered. The remaining diffusely involved mesentery was rubbery, firm, mottled, opaque and non-greasy, and had a variable greyish white to yellowish brown color. Its cut surface showed multiple yellowish orange nodules varying from 1 to several centimeters in diameter. Many of these nodules displayed a small greyish lymph node centrally and a surrounding zone of relatively normal fat. The lymph nodes themselves were not enlarged or ectatic and there was no evidence of lymphatic obstruction. No cysts or granulomatous foci were seen grossly. These mesenteric tumors were confined solely to the small bowel mesentery in all cases and the intestine proper showed no abnormalities. The remaining abdominal mesenteries and omentum, as well as the perivisceral and retroperitoneal fatty tissues, were unaltered, and the liver and pancreas were normal.

Microscopically, the mesenteric tumor masses were composed of nodules of various size made up of non-neoplastic but structurally altered fat cells. Some of the smaller nodules were comprised entirely of these abnormal cells. Many of the larger, more deeply situated nodules exhibited a characteristic peripheral distribution of these cells. (Fig. 3.) In the center of the nodules there was usually a small, relatively normal lymph node or blood vessel, about which was a zone of normal fat. These cores were collared by a wide peripheral zone of abnormal fat cells, corresponding to the opaque, pale yellowish grey areas described grossly. These same cells were present as a broad layer beneath the peritoneal surface of the mesentery. (Fig. 4.)

The abnormal fat cells were round to oval and were supported by a delicate reticular framework. They varied considerably in size, most being much smaller than normal. (Fig. 5.) The cytoplasm, with routine staining, was faintly basophilic and finely granular. (Fig. 6.) Occasionally within the cytoplasm there were one or more small clear vacuoles, presumably representing neutral fat. The nucleus tended to be centrally placed, small, round and hyperchromatic. Frequently, more than one nucleus could be identified within the limits of a single cell membrane. With frozen sections, Oil Red O stained the normal fat cells homogeneous deep

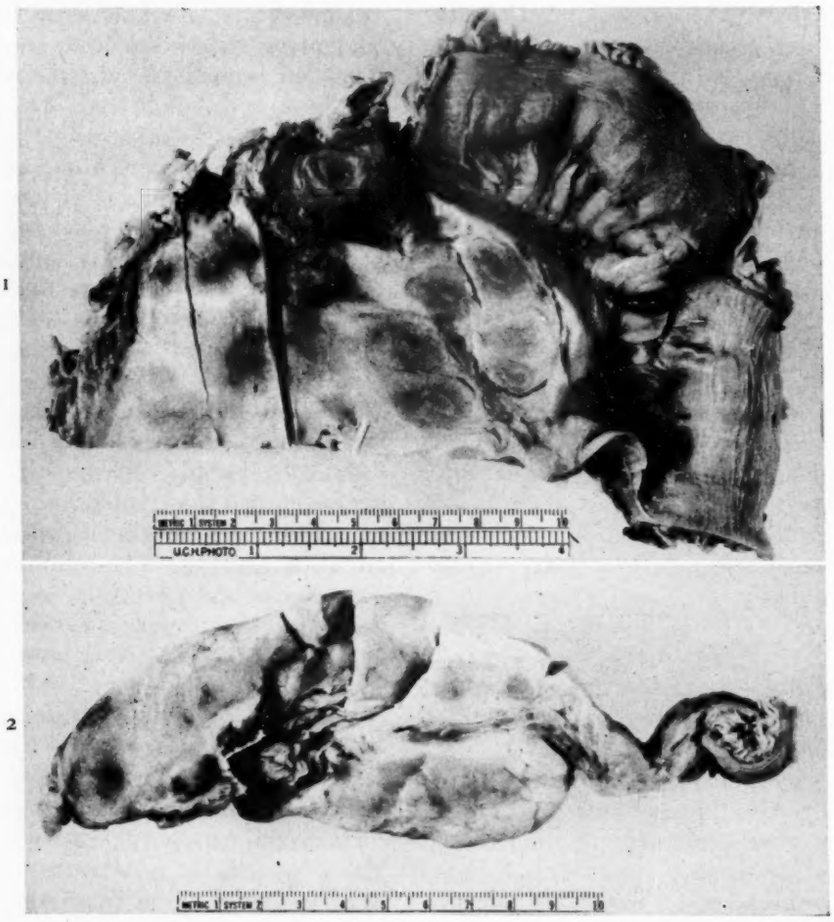

FIG. 1. Case 1. The nodularity of the mesenteric mass is shown in a mid-sagittal section. Several of the nodules have a small centrally placed lymph node surrounded by a zone of relatively normal fat (dark grey). Beyond this area and between nodules is much rubbery, firm, non-greasy, mottled, abnormal fat tissue (light grey). A portion of the small intestine is attached (upper right).

FIG. 2. Same case. One-half the thickness of the mesenteric mass is represented on cross section. The abnormal fatty tissue is particularly prominent as a broad zone immediately beneath the peritoneal surface of the mesentery. Note that the portion of the mesentery adjacent to the small intestine (far right) is relatively uninvolved.

red. With deviation from normal, this sudanophilia decreased and, as progressively diminishing small vacuoles, finally disappeared. At the same time, progressively accumulating intracytoplasmic crystals appeared. Initially they were delicate, needle-like structures, visible with reduced light and occasionally anisotropic. Later they tended to coalesce and coarsen and to extrude through the cell membrane. The process in four of the cases did not progress beyond this point. In the fifth case, however, it was with this focal liberation of crystals and coincident breakdown of fat cells that the initial inflammatory changes were observed. (Fig. 7.) Small, widely separated infiltrates of monocytes and lymphocytes appeared, followed by macrophages, rare foreign body giant cells and later fibroblasts. Only rarely did this inflammatory reaction progress to central tissue necrosis with marked extracellular crystalline deposition and active lipophagocytosis, fibrous proliferation and sclerosis, i.e., lipogranulomatosis. (Fig. 8.)

From a therapeutic and prognostic standpoint, mesenteric lipodystrophy must be differentiated from lipoma, liposarcoma, fibrosarcoma, lymphosarcoma, mesenteric lymphadenitis, Whipple's disease, Weber-Christian disease and sclerosing lipogranuloma.

Lipomas, among the most common solid tumors of the mesentery, may be bulky and nodular but appear well encapsulated. Although

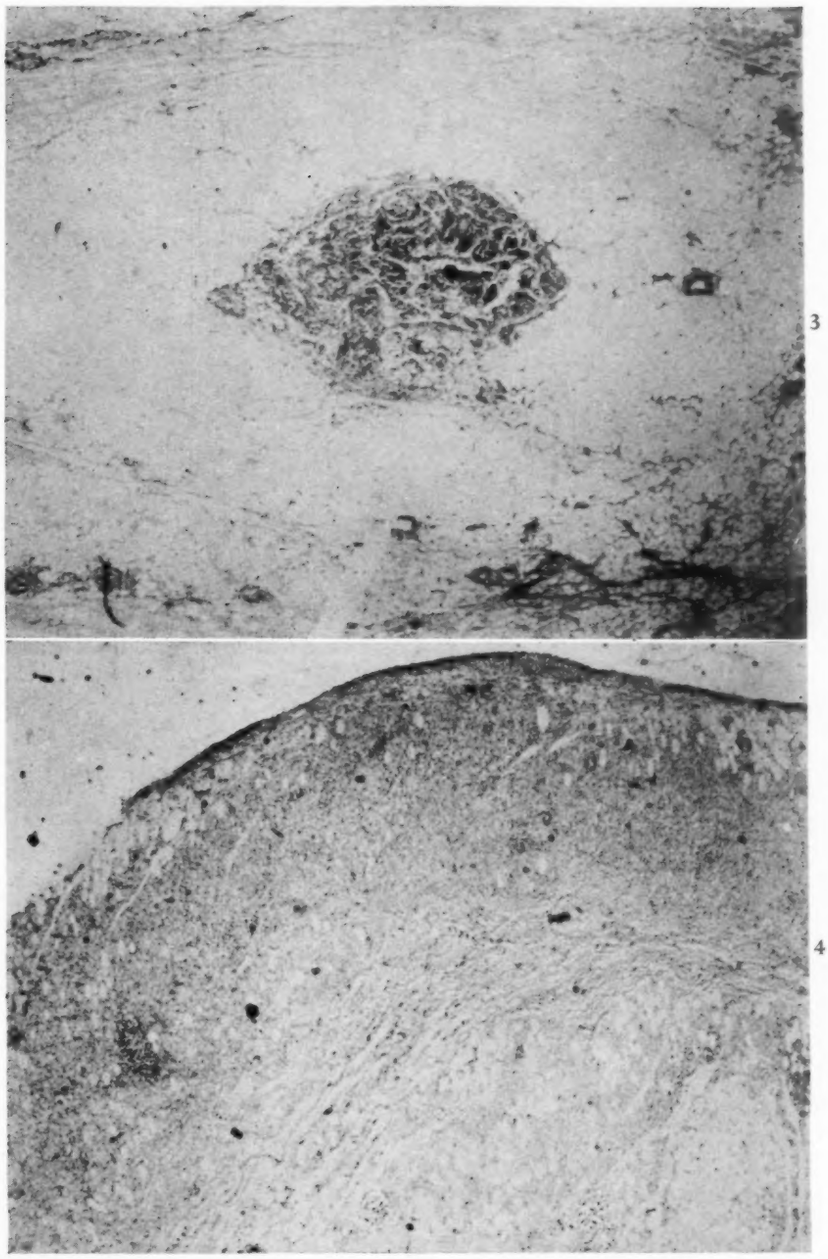

FIG. 3. Case V. A small mesenteric nodule, demonstrating the "halo" effect. A small, centrally situated lymph node is surrounded by a core of normal fat cells (clear). The peripheral portion of the lobule is comprised of abnormal fat cells (opaque).

FIG. 4. Case III. The localization of abnormal fat cells in a broad zone beneath the peritoneal surface of the mesentery is shown. Note that there is no tissue necrosis and only a few small groups of lymphocytes (far left).

most often soft and bright yellow, they become increasingly rubbery firm and greyish as their fibrous tissue component increases. The lipoma cells have the same microscopic and chemical characteristics as normal adult fat cells. There may sometimes be areas of fat necrosis centrally.[1,3,9,10,19,25] Multiple, familial or congenital lipomatosis, including such conditions as Dercum's disease, do not pose a sufficient differential diagnostic problem to warrant discussion here.[7,22,23]

Of the primary malignant tumors which may involve the mesenteric area, liposarcoma and fibrosarcoma are the most important.[8,24,27,35]

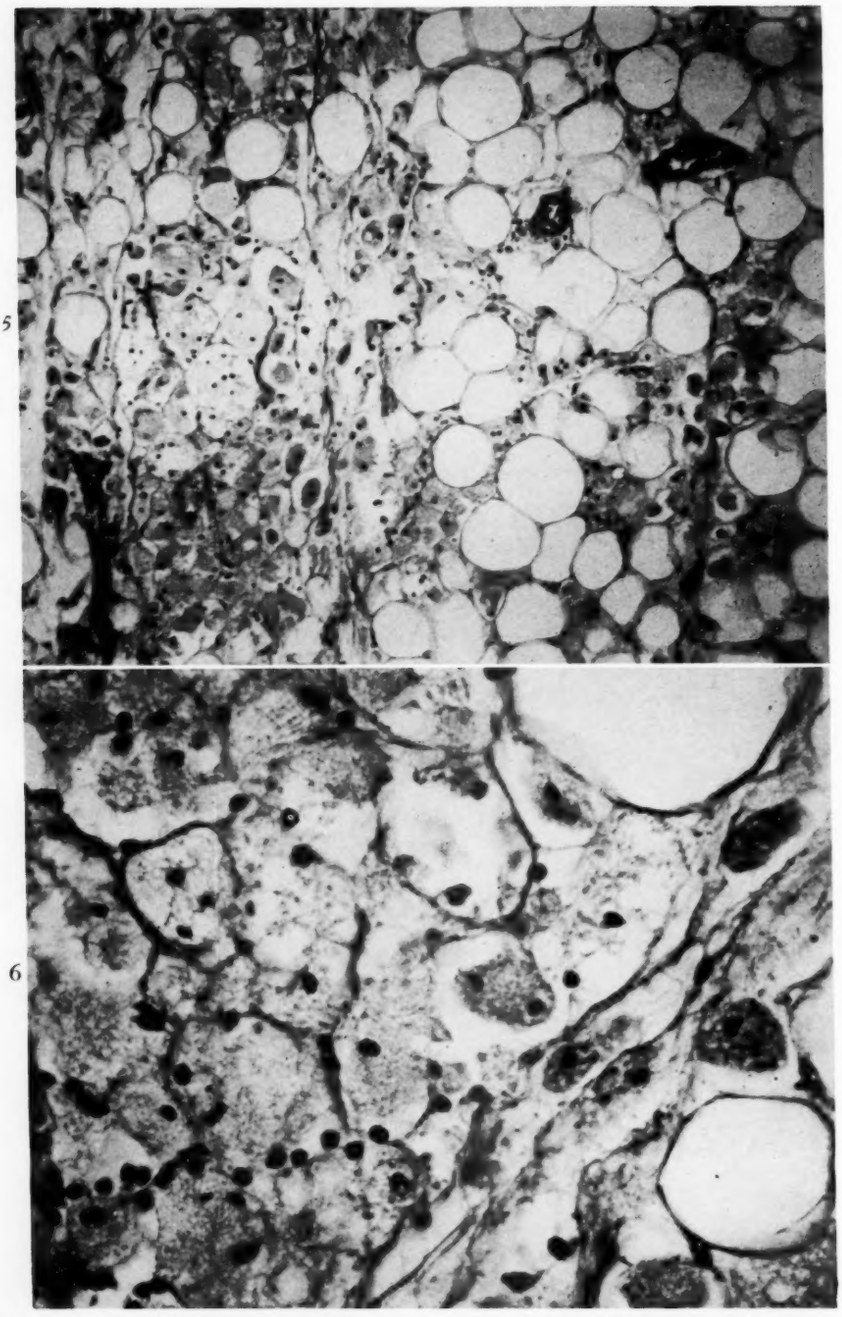

FIG. 5. Case IV. Normal fat cells are interspersed between numbers of altered fat cells. The latter are smaller than normal with opaque cytoplasm and small, centrally situated nuclei. Note that there are no inflammatory cells or phagocytes and that the small vessels are normal (upper right and lower left).

FIG. 6. Same case. The fine granularity and the occasional clear vacuolization of the altered fat cell cytoplasm are shown at higher magnification. Many of these cells apparently contain more than one nucleus. Two relatively normal fat cells are present (upper and lower right).

By the time they are palpable clinically they usually give signs of organ displacement or obstruction. Grossly bulky, firm and sometimes nodular, they are usually tan to dull grey and show surface mottling. Microscopic examination of biopsy tissue at the time of operation usually will indicate the malignant nature of these tumors.

Lymphosarcoma commonly involves the mesenteric lymph nodes and may simulate

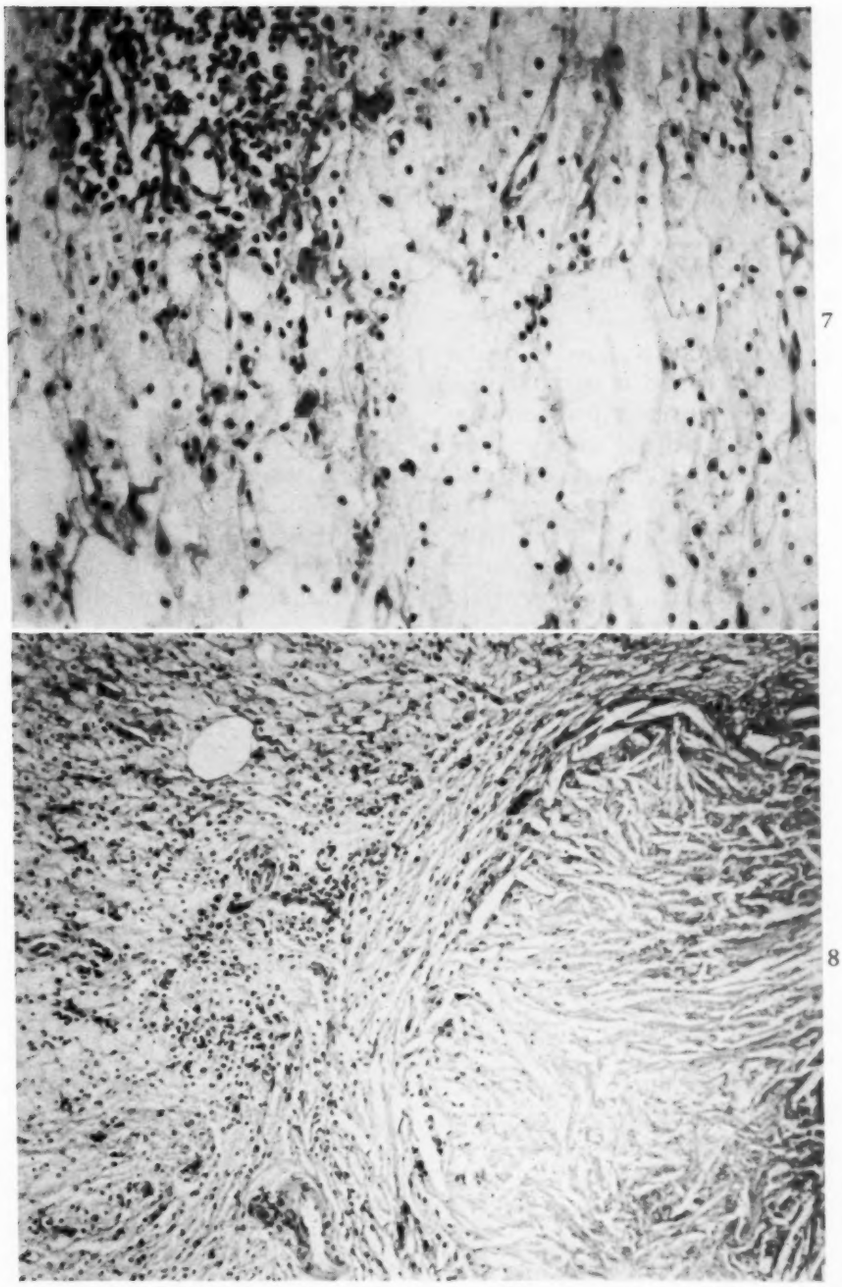

FIG. 7. Case 1. A small infiltrate of mononuclear cells (upper left) is present in a broad field of abnormal fat cells. Focal collections of lymphocytes and phagocytes probably indicate the initial inflammatory reaction to the degenerating fat.

FIG. 8. Same case. A focus of "lipogranulomatous inflammation" (right) is present as a discrete nodule in a large field of abnormal fat cells. Centrally, the nodule is comprised of cellular debris and acicular clefts (crystalline material) about which is a collar of phagocytes, giant cells and lymphocytes.

nodular lipodystrophy.[12] There may or may not be associated chylous ascites. The correct diagnosis is made by exposing the mesenteric lymph nodes at operation and removing one for microscopic examination.

Mesenteric lymphadenitis, particularly when caused by tuberculosis or sarcoid, may appear as a mesenteric mass. Here, as in lymphosarcoma, surgical exposure, biopsy and histologic examination of the mesenteric lymph nodes will lead to establishment of the diagnosis.

Of the fifty odd cases of Whipple's disease

reported, there are a few in which the diagnosis is questioned. Certain of these, while perhaps representing true examples of Whipple's disease, lack sufficient clinical or pathologic data to substantiate this diagnosis.[4,5] Others, in all probability, are identical to our cases and could be classified as examples of "mesenteric lipodystrophy."[30–32]

Whipple's disease is a chronic, slowly progressive illness characterized by diarrhea, weight loss, emaciation and, ultimately, death.[20,36] Quite frequently there are associated arthritis and serositis. Steatorrhea and a peculiar pigmentation of the skin are usually present. The generally recognized pathologic changes are macrophagocytosis of the bowel mucosa, chylangiectasis and lipogranulomatosis of the mesenteric lymph nodes, and dilatation of the mesenteric lymph vessels.[34] Chylous ascites may or may not be present. The mesenteric fat shows only minimal changes with atrophy of fat cells and mild focal lymphocytic infiltration. Fat necrosis is notably absent.

In contrast, "mesenteric lipodystrophy" is a disease remarkable in its paucity of signs and symptoms; an ill defined abdominal mass is the only consistent finding. Pathologic change is confined to the mesenteric fat; the bowel proper, mesenteric lymph vessels and lymph nodes are relatively uninvolved.

Pathologic differentiation between Whipple's disease and primary mesenteric lipodystrophy depends upon histologic interpretation of the bowel proper and mesenteric lymph nodes, as well as the mesenteric fat.

Relapsing febrile non-suppurative panniculitis (Weber-Christian disease) is characterized by the development of successive crops of inflammatory nodules in subcutaneous fat; these heal by fibrosis, leaving shallow cutaneous depressions.[11,16,21,28,33,38,42] Exacerbations are accompanied by fever and malaise, while remissions are relatively asymptomatic. In the majority of cases reported the disease is limited to the subcutaneous panniculus. In some, however, there is generalized involvement including the visceral, omental and mesenteric fat as well as other adipose sites.[14,26,40,41]

Lesions of Weber-Christian disease are confined to fat lobules and are characterized by a granulomatous inflammation, with collections of leukocytes, lymphocytes, plasma cells, fat-laden macrophages and occasional multinucleated giant cells centered about areas of fat necrosis. Various amounts of fibrosis are described. Intimal proliferation and hyalinization of small blood vessels as well as perivascular inflammation are frequently observed.

In contrast, in mesenteric lipodystrophy, fever and malaise as well as other constitutional symptoms of Weber-Christian disease are absent. Systemic fat deposits, especially the subcutaneous panniculus, are never affected. Although the small bowel mesentery in systemic Weber-Christian disease may closely resemble those of our cases in respect to gross features, there are basic microscopic differences. In the former inflammation overshadows and apparently precedes degeneration of fatty tissue, while in the latter inflammatory changes are minimal and most probably secondary.

Primary granulomatous reactions of fat (lipogranulomas) generally are attributed to some form of injury and most often are subcutaneous in location.[2,6,15,17,18,37] They form irregular or nodular tumorous masses and are firm, opaque and yellowish to greyish white. They often contain small cysts (disrupted fat cells which have coalesced). Macrophages and giant cells invariably are present, as well as collections of crystals or cleft-like spaces and proliferating fibrous tissue. The peripheral portions of the involved fat lobules often show a peculiar necrosis with hyalinization and scarring.

Oberling[29] reported the occurrence of three large, firm, greyish yellow retroperitoneal tumors which he called xanthogranulomas. Histologically, these masses were composed of inflammatory tissue infiltrated extensively by foam cells filled with droplets which stained intensely with Scharlach R. Some also contained sheaves of needle-like anisotropic crystals. Although these cases represented predominantly a granulomatous process, it appears from Oberling's illustrations that in addition to the histiocytes and giant cells there were large numbers of altered fat cells. Oberling stated that the etiology and pathogenesis of xanthogranuloma are unknown.

However, xanthogranuloma, pancreatic and traumatic fat necrosis,[43] and sclerema neonatorum[13] all closely resemble sclerosing lipogranuloma. Most of these conditions have known etiologic agents, i.e., enzymatic activity and/or trauma. The term "traumatic fat necrosis," while nonetheless valid, is often overapplied. Spenser[39] pointed out that al-

though the characteristic granulomatous process frequently follows mechanical injury to fat, identical lesions develop in about 50 per cent of patients in whom no history of trauma can be elicited. Since it is recognized that human fat is unstable and breaks down easily into fatty acid and glycerol, it is conceivable that a variety of etiologic factors might cause the granulomatous change so commonly labeled "traumatic fat necrosis." In none of our cases was there evidence of trauma or pancreatic disease. In examples of pancreatic fat necrosis involving the mesentery the reaction is superficial and does not produce a mesenteric mass.

The large group, known variously as xanthogranuloma, lipogranuloma, fat necrosis and so forth, represents a pathologic process closest in over-all character to mesenteric lipodystrophy. The gross pathology may be strikingly similar, although no cases have been reported involving only the mesentery. The chief difference is the marked disparity in degree of granulomatous inflammation in the two processes. In the lipogranulomas it predominates; in mesenteric lipodystrophy it is inconspicuous. The similarities are the disruption of fat cells the presence of crystalline material within the cytoplasm of the altered fat cells and the preponderant accumulations of these abnormal cells about the periphery of fat lobules, farthest removed from the central nutrient arterioles. These parallels in evolution of the two disease processes, however, do not necessarily indicate a common underlying etiology.

In nodular mesenteric lipodystrophy any interpretation of the etiology and early pathologic changes should take into consideration and correlate the following basic findings: (1) The mesenteric mass results from the increased size of pre-existing fat lobules. (2) Altered, degenerating and necrotic fat cells are largely confined to the periphery of these lobules. (3) The altered fat cells apparently do not contribute to the bulk of the tumor, as they are smaller than normal fat cells and lack evidence of proliferation. (4) Marked tissue destruction, inflammation and vascular lesions are never present in the early stages.

It is postulated that the initial change in nodular mesenteric lipodystrophy may well be a non-neoplastic proliferation of normal fat cells in the central portions of lobules throughout the mesentery. As this process progresses, fat cells at the periphery of the lobules are compromised through either vascular or mechanical embarrassment. An alteration in intracytoplasmic fat metabolism results and encompasses faulty absorption of circulating molecular fat, uninhibited hydrolysis of neutral fat to fatty acids and glycerol, incomplete oxidation of fatty acids and intracellular saponification of these acids.

In most cases the process, although slowly progressive, does not pass beyond this stage. Occasionally, however, actual focal breakdown of fat cells, liberation of fatty acid crystals and resultant lipogranulomatous inflammation occur.

SUMMARY

A peculiar tumor-like alteration of the fat of the small bowel mesentery is described. This lesion was found in five adult patients, all with little or no relating symptomatology. In four an abdominal mass was palpable. Diagnosis was established at exploratory laparotomy in three patients, and at postmortem examination in the remaining two.

Grossly, the entire small bowel mesentery was thickened and nodular, rubbery firm, opaque and mottled grey to yellowish orange.

Microscopically, there was diffuse involvement of fat cells, most prominent about the periphery of the fat lobules. In the early stages individual fat cells appeared smaller than normal; their cytoplasm was opaque, granular and faintly basophilic, and the nucleus was centrally situated. Later, a mild focal lymphocytic infiltration and perilobular condensation of fibrous tissue were apparent. Only rarely and in small focal areas was there development of lipogranulomas.

It is suggested that initial proliferation of mesenteric fat induces a perpetuating alteration in fat cell metabolism through either vascular or mechanical embarrassment. The fat cells, particularly those of the relatively more ischemic peripheral portions of the lobules, lose their ability to absorb fat and their lipolytic activity is compromised. These altered cells undergo the structural changes described heretofore; and as the process progresses, they may actually break down, liberating fatty acids and exciting focal inflammation and fibrosis.

These mesenteric masses bear certain resemblances to Whipple's disease, Weber-Christian disease with systemic involvement,

sclerosing lipogranuloma and traumatic or pancreatic fat necrosis. They must also be differentiated from true fat tumors, lymphosarcoma, tuberculosis and other less common conditions. The premortem diagnosis can be made only at operation. Biopsies should be taken not only of the mesentery but also of the bowel and mesenteric lymph nodes. Surgical resection usually is not feasible, as the process tends to surround the major mesenteric vessels. The prognosis apparently is favorable, for the lesion is slowly progressive and probably self-limited. Alteration in bowel function does not ensue, nor is there evidence that the process later involves other tissues.

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DISCUSSION

Jack Matthews Farris (Los Angeles, Calif.): After discussing this problem with a good number of my friends in Los Angeles, I was able to uncover a case which I am sure is identical with those described by Drs. Crane, Aguilar and Grimes. The details of this case were related to me by Dr. H. Russell Fisher, who is the pathologist of the Huntington Memorial Hospital in Pasadena.

This patient was an adult woman who made a habit of submitting to numerous examinations at various cancer detection centers. During the course of one of these examinations someone discovered a tumor in her abdomen which was completely asymptomatic. Likewise, an exhaustive diagnostic study failed to reveal the exact nature of the lesion and she therefore underwent operation. There was an enormous accumulation of odd-looking lobulated fat in the mesentery of the small bowel surrounding the root of the mesentery. The lobules showed a yellow color identical with the lesions shown by the authors. Dispersed through these lobules were areas of chalky fat necrosis. Microscopically, the fat cells were smaller than normal and demonstrated the same basophilic thickening and reticular network which I am sure is identical with the microscopic pattern illustrated in these cases. Although at first the lesion was thought to be a liposarcoma or some similar tumor, a biopsy examination was carried out at the operating table and the true nature of the lesion thereby ascertained. Fortunately, resection of the bowel and the mesentery was not performed.

I am sure it is apparent to everyone after reviewing the authors' cases that resection when undertaken will involve removal of the entire small bowel and frequently part of the ascending colon because of the involvement about the vascular root of the mesentery. One characteristic of this distribution of fat is its tendency to recede toward the periphery of the mesentery without any involvement of the bowel. The lymphatics are not involved and the blood vessels appear to be normal as well.

Fatty substances form a considerable portion of all cells and tissues, and disturbances in amounts, distribution and types and stainability are common pathologic features of many diseases. It seems to us that perhaps these accumulations of fat in the mesentery of the small bowel may well be analogous to the so-called retroperitoneal lipoma, which is notorious for its ability to dissect other retroperitoneal structures; for example, the retroperitoneal lipoma which surrounds the pedicle of the kidney, and the enormous ones which may occupy the subphrenic space, again without involvement of vascular trunks. Inflammatory changes occurring in these abnormal accumulations of fat, related either to trauma or impaired vascular supply, may produce areas of focal necrosis and a pathologic picture which is similar to the one shown by the authors. Other abnormal accumulations are seen in the so-called "hibernoma," a depot of odd-appearing fat at the base of the cervical spine. Unusual accumulations of fat have likewise been noted in the buttocks of certain Hottentots and Bushmen. Perhaps these accumulations of fat in the mesentery may bear some remote relationship.

I am extremely grateful for the opportunity to discuss this important paper, and I sincerely hope that when I am confronted with such a lesion I will be able to recognize it, biopsy it and refrain from resection of the small intestine.

[The following questions were asked from the floor and answered by Dr. Jackson T. Crane.]

Joseph A. Weinberg (Long Beach, Calif.): Are there any signs or symptoms peculiar to this disease other than a palpable mass?

Answer: I am sure a palpable mass is the only constant finding that is present. On x-ray study there usually is no displacement of the kidneys. The bowel may be pushed forward so that a portion of the duodenum may appear to lie farther anterior than it should normally.

Allen M. Boyden (Portland, Ore.): Did any of these patients have a history of an acute abdominal episode in the past such as might be attributable to pancreatitis?

Answer: Two of the patients had their appendix removed for acute appendicitis several years prior to the known development of the tumor. No remarks were made at that time as to the condition of the mesentery of the small bowel. We have autopsy material on two cases with no evidence of pancreatitis, either acute or chronic. The remaining three patients, all living, show no evidence of pancreatic dysfunction.

Clinical Evaluation of Bilateral Adrenalectomy and Oophorectomy for Advanced Mammary Carcinoma

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From the Division of Surgery, the Cancer Research Institute and the Metabolic Unit, University of California School of Medicine, San Francisco, Calif.

SINCE 1946 patients with symptoms from advanced mammary carcinoma have been treated in the breast clinic of the University of California Hospital with hormone therapy when the disease extended beyond the possibility of treatment with surgical or radiation methods. Hormonal therapy may produce a remission of symptoms in suitable cases until a favorable response to the administration of either androgens or estrogens can no longer be obtained. The work of Huggins demonstrated that an additional temporary remission may occur when bilateral adrenalectomy and oophorectomy is performed in selected patients. This procedure was carried out in an attempt to remove all major sources of estrogen in patients with mammary cancer presumably still retaining the parent-tissue sensitivity to estrogenic substances. Following a visit and demonstration by Huggins in 1952, several patients with advanced breast cancer were selected for bilateral adrenalectomy and oophorectomy. This report is concerned with a clinical evaluation of the procedure in fifty patients.*

Selection of Patients for Operation. Only patients with objectively demonstrated breast cancer advanced beyond the possibility of further treatment with surgery or radiation were selected for adrenalectomy and oophorectomy. (Table I.) Usually some form of hormone therapy was tried, and the patient had either failed to respond or had escaped hormone control of the breast cancer before adrenalectomy was advised. Most of these patients had widespread disease, usually in one or more of the following locations: osseous, pulmonary, pleural, abdominal viscera, lymphatics and/or local recurrence in the chest wall. The operations frequently were performed on patients confined to bed with debility and pain, but usually not on those apparently moribund; it probably is not indicated in patients jaundiced from extensive liver metastases. The age of these patients varied from twenty-eight to

TABLE I

CRITERIA FOR SELECTION OF PATIENTS FOR ADRENALECTOMY AND OOPHORECTOMY

1. Age (advanced age not a contraindication)
2. Metastases demonstrated objectively
3. Symptoms present (usually severe pain)
4. Standard therapy (surgery, radiation, hormones) no longer indicated
5. Location of lesions: bones, soft tissues, serous cavities
6. Histologic pattern: adenocarcinoma preferable but not necessary

TABLE II

ADRENAL REPLACEMENT THERAPY

Day	Cortisone Acetate	Blood Pressure	DOCA
Preoperative	100 mg. intramuscularly night and 1 hr. before operation	Three determinations	.....
First.....	50 mg. intramuscularly every 6 hr.	Every 15 min.	.....
Second.....	50 mg. orally every 6 hr.	Every hr.	.....
Third.....	25 mg. orally every 6 hr.	Every 2 hr.	2 mg., if no evidence of heart failure is present
Fourth.....	25 mg. orally every 8 hr.	.....	1 mg. daily intramuscularly
Fifth.....	12.5 mg. orally every 8 hr.	.....	1 mg. daily intramuscularly
Sixth and on	12.5 mg. orally every 6 to 8 hr. depending on requirement	.....	25 to 50 mg. trimethylacetate of desoxycorticosterone intramuscularly as required every 21 to 30 days

* One patient was operated upon in Chicago in 1952 by Dr. C. Huggins, to whom we are indebted for her follow-up in our clinic.

seventy-two years. Advanced age did not constitute a deterrent if the procedure was indicated and the patient was judged able to survive the operation.

In this series no correlation was found between the clinical response and adrenal gland weight (as determined preoperatively) or adrenal functional response (as studied preoperatively by maximal stimulation with intravenous corticotropin). All patients whose primary lesion consisted of a glandular pattern (adenocarcinoma) showed a good clinical response. A considerable number of those with histologic patterns devoid of glandular organization showed equally good results. Thus these criteria could not be accepted as valuable guides for the proper selection of patients for the operation.

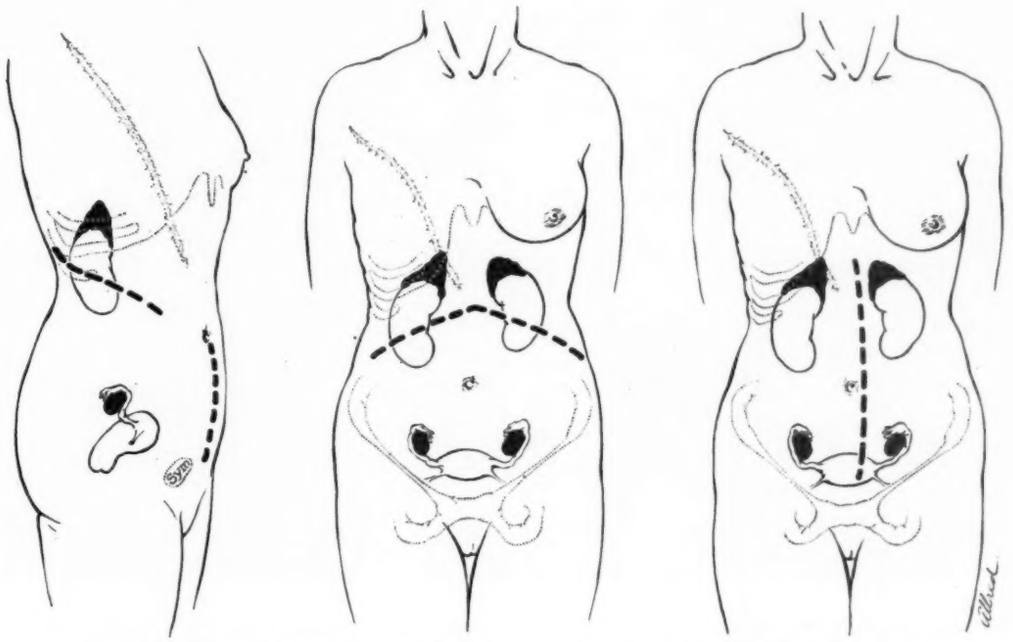


FIG. 1. Diagram of incisions used for the removal of the adrenal glands and ovaries.

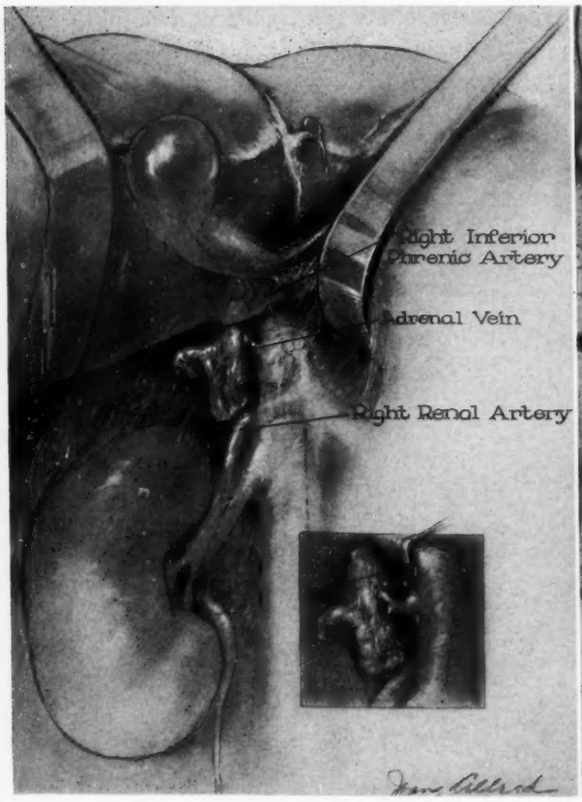
Preoperative Preparation. Usually patients were hospitalized two to four days prior to operation. In addition to routine laboratory work as for any other major surgical procedure, calcium, phosphorus and basic phosphatase blood levels were determined, which were to serve as subsequent laboratory indications of the course of the disease. Complete bone surveys were obtained preoperatively and at regular intervals postoperatively. The plan utilized for adrenal replacement therapy is given in Table II.

Because of its relatively slow solubility and absorption cortisone acetate was given intramuscularly on the evening prior to operation and on the morning of operation in doses of 100 mg. each. The other alternative is to utilize intravenous hydrocortisone concentrate (100 mg. in 50 per cent alcohol added *in toto* to 500 or 1000 ml. of 5 per cent dextrose in water). This infusion is started at a moderate speed at the time of incision without any previous special preparation, and is followed throughout the first postoperative day by another 1,000 ml. containing 100 mg. of hydrocortisone. The following day another 200 mg. are administered, or the patient is given cortisone acetate intramuscularly from the first day on. (Table II.)

A No. 18 or larger needle is always introduced into an ankle vein by means of a cutdown prior to the induction of anesthesia. The utmost care is exercised to ascertain proper functioning of this cutdown at all times so that blood, hydrocortisone, or other drugs can be given properly and rapidly when indicated.

A catheter is left in the urinary bladder for accurate measurement of urinary output every six hours.

Operation. Endotracheal inhalation anesthesia was used for most operations, utilizing various agents (ether, cyclopropane, nitrous



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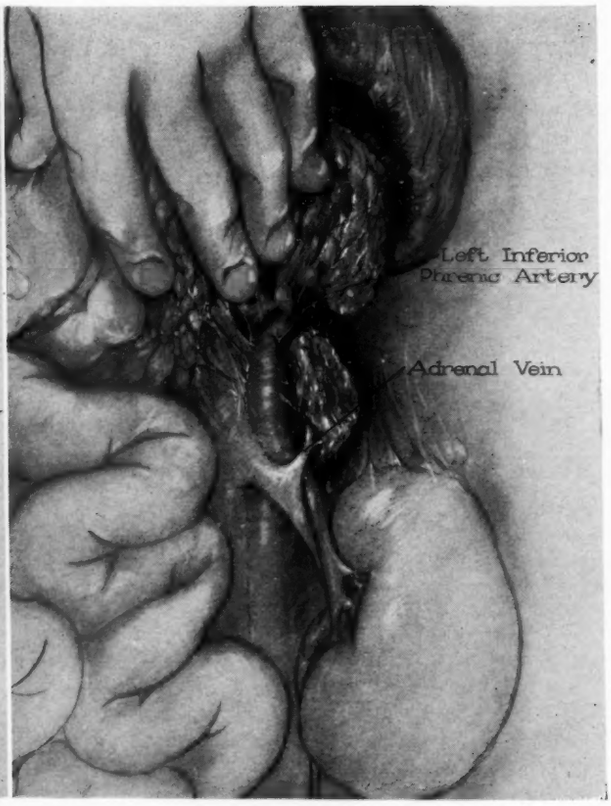

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FIG. 2. Drawing showing exposure of the right adrenal gland. Note its high position retroperitoneally; the apex of the gland is usually behind the liver.

FIG. 3. Drawing showing exposure of the left adrenal gland. Note the mobilization of the pancreas anteromedially for removal of the gland which partially occupies a retropancreatic position.

oxide, etc.). Spinal anesthesia was employed occasionally. Patients undergoing adrenalectomy tolerate barbiturates poorly, and their use has been omitted altogether in poor risk patients.

Hydrocortisone (free alcohol), blood and methoxamine (vasoxyl®) should be on hand in the operating room in adequate amounts at all times.

Figure 1 illustrates the various approaches utilized for removal of the adrenal glands and the ovaries. Bilateral flank incisions as described by Huggins were used in twenty-three cases. In these patients oophorectomy was performed at the same sitting through a third lower midline incision, adrenalectomy and oophorectomy usually being performed in one stage unless both ovaries had already been removed. In one case oophorectomy was performed at a later stage, three weeks after adrenalectomy. Twenty patients were operated upon through an inverted curvilinear transverse upper abdominal wound, and seven through a long paramedian incision. The transabdominal approach is easier on the patients and is preferable in cases with extensive spinal metastases as it decreases the possibility of compression fracture during turning and positioning of the patient. However, removal of the adrenal glands, particularly the right one, is considerably more difficult through the laparotomy incision as compared to the relatively easy adrenalectomy through flank incisions. The laparotomy incision enables the operator to search for accessory adrenal tissue which has been reported to occur in up to 20 per cent of random subjects. (Fig. 4.)

The adrenals should be approached carefully by either method in order to remove them intact. If the adrenals are torn or fragmented, incomplete removal may result and the patient may lose the possible beneficial effects of the procedure. The several arteries are quite small and usually are controlled with the application

of hemostatic clips. The adrenal veins are considerably larger and should be secured with ligatures. (Figs. 2 and 3.)

Postoperative Care. This consists essentially of the usual measures employed in the proper administration of fluids, electrolytes and sedatives used during any major surgical procedure. Gastric and intestinal aspiration is maintained through an indwelling catheter until active peristalsis is present.

From the first day on, adrenal replacement therapy is employed as outlined in Table II. Barring complicating factors, the patient is placed on a maintenance dose of 12.5 mg. of cortisone acetate every six to eight hours orally by the fifth day. Since it is necessary to supplement the somewhat insufficient sodium-retaining and blood pressure–stabilizing action of this maintenance dose of cortisone acetate, the patient is given 25 to 50 mg. of trimethyl acetate of desoxycorticosterone every twenty-one or thirty days, regulated so as to maintain adequate hydration and weight on a normal diet. Sodium intake should be the usual 6 to 8 gm. per day. In a few patients supplementary sodium chloride may be required and is given as enteric-coated pills (1 gm. three times daily). This obviates the necessity of administering larger amounts of cortisone acetate which is likely to produce excessive weight gain, rounding of the face, insomnia and other undesirable effects.

Careful management of replacement therapy is an essential part in the care of adrenalectomized patients and is imperative for the survival of patients following adrenalectomy. This procedure should be undertaken only when adequate facilities for proper electrolyte determinations and substitution therapy are available.

Follow-up. Adrenalectomized patients must be instructed carefully as to their physiologic status. The following printed form is given to the patient on discharge and has proved most useful:

LIFE WITHOUT ADRENALS

"Without your adrenals it is absolutely essential that you continue to take cortisone for the rest of your life. The amount that you will require may vary from time to time, e.g., if you have an illness of any sort you will require more. You are therefore advised to see your doctor on all occasions of even minor illnesses.

"The injections you have been receiving in the hospital are desoxycorticosterone acetate (DOCA) and before discharge you received a special injection of this substance which will last for one month. This helps to retain a normal amount of salt in your body and keeps your blood pressure up to normal. The injection must be repeated every month.

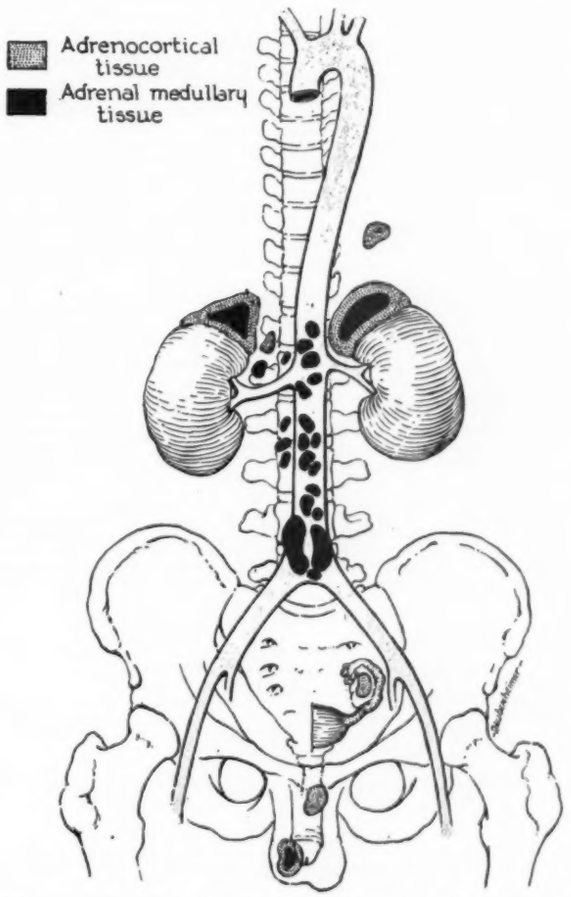

FIG. 4. Diagram showing usual locations of accessory adrenocortical and medullary tissue, if present. (From FORSHAM, P. H. and THORN, G. W. The adrenals. In: Williams' Textbook of Endocrinology. Philadelphia, 1955. W. B. Saunders Co.)

"We have also found that it is necessary for you to take extra salt. In order to be sure that you are getting the correct amount of salt it is important that you should weigh yourself at the same time every day, preferably before breakfast. If, over a period of 2 or 3 days, your weight increases rapidly, this means that you are getting too much salt and you should reduce the dose, e.g., from 2 tablets to 1 tablet. Similarly, if your weight falls over a period of

TABLE III

RESULTS OF BILATERAL ADRENALECTOMY AND OOPHORECTOMY*

Patient	Age (yr.)	Menopausal Status	Time since Mastectomy	Location of Metastases	Other Treatment	Survival since Adrenalectomy	Objective Response	Subjective Response
R. V. W.	44	3 yr. postmenopausal	3½ yr.	Adrenals, ovaries, osseous	Radiation to ovaries and bones; androgens	10 mo. (died)	Progression of lesions	Cessation of pain
G. C.	42	Premenopausal	1 yr.	Liver, brain, ovaries, adrenals	Physiotherapy	7 mo. (died)	Scotoma reduced	Cessation of pain
M. D.	60	18 yr. postmenopausal	9 mo.	Pulmonary, pericardial	Estrogens; androgens	16 mo. (died)	Progression of metastases	Cessation of pain
M. G.	67	10 yr. postmenopausal	10 yr.	Chest wall ulcerations, lymph nodes	Radiation; androgens	9 mo. (died)	Healing of ulcers	Cessation of pain
V. H.	60	14 yr. postmenopausal	8 yr.	Lymph nodes, osseous	Radiation; estrogens	18 mo.	Recalcification of osteolytic lesions	Cessation of pain
E. H.	43	4 yr. postmenopausal	4 yr.	Lymph nodes, skeletal	Radiation; androgens	6 mo. (died)	Recalcification of osteolytic lesions	Cessation of pain
R. H.	50	Radiation menopause after mastectomy	4 yr.	Liver, lymph nodes	Androgens	2 mo. (died)	Progression of metastases	None
R. E. H.	40	5 mo. postmenopausal	6 yr.	Osseous, pleural effusion	Radiation; androgens	28 mo.	Recalcification of osteolytic lesions	Cessation of pain; ambulatory; working
E. J.	57	8 yr. postmenopausal	3 yr.	Pulmonary, pleural, osseous		11 mo. (died)	None	None
I. M.	36	1½ yr. postmenopausal	2 yr., 9 mo.	Osseous, pulmonary	Radiation; androgens	2½ mo. (died)	Progression of lesions	None
M. N.	57	5 yr. postmenopausal	5½ yr.	Osseous	Radiation; androgens	3 mo. (died)	None	None
J. P.	53	4 yr. postmenopausal	5 yr., 7 mo.	Osseous	Radiation; androgens	8 mo. (died)	None	None
V. P.	41	1 mo. postmenopausal (surgical)	2 yr.	Osseous, lymph nodes, liver	Estrogens; androgens	1 mo. (died)	None	None
M. D.	49	6 yr. postmenopausal (surgical)	6½ yr.	Osseous	Radiation; androgens	9 mo. (died)	None	None
D. S.	53	1 yr. postmenopausal (radiation)	10½ mo.	Lymph nodes, soft tissue	Ovarian ablation by radiation	14 mo. (died)	None	None
E. S.	56	8 yr. postmenopausal	2 yr., 5 mo.	Chest wall, soft tissue	Radiation; estrogens	6 mo. (died)	Progression of lesions	None
V. S.	37	5 yr. postmenopausal (surgical)	9 mo.	Osseous, lymph nodes, soft tissue, adrenals	Radiation; androgens	16 mo. (died)	Progression of lesions	Relief of symptoms
S. W.	53	Postmenopausal	5 yr.	Skin, osseous, lymph nodes	Radiation; androgens	18 mo.	Recalcification of osteolytic lesions	Cessation of pain
L. B.	62	19 yr. postmenopausal	8 yr., 8 mo.	Osseous, lymph nodes	Radiation	13 mo.	Increase in osseous metastases	Relief of pain
M. McD.	72	2 yr. postmenopausal	2 yr.	Lymph nodes, soft tissue	Radiation	7 mo. (died)	Progression of lesions	None
I. C.	33	Premenopausal	4 yr., 7 mo.	Lymph nodes, skin, contralateral breast	Radiation	2 yr.	Regression of breast lesion; healing of ulcerations	Patient asymptomatic prior to adrenalectomy
L. S.	28	3 mo. postmenopausal (surgical)	1 yr., 4 mo.	Lymph nodes, contralateral breast, skin	Radiation	10 mo.	Progression of lesions	None
G. M.	33	Premenopausal	7 mo.	Osseous, pulmonary	Radiation; androgens	Died	None	None
F. A.	44	Premenopausal	†	Pulmonary, osseous	Androgens	1 mo. (died)	None	None

TABLE III (*Continued*)

Patient	Age (yr.)	Menopausal Status	Time since Mastectomy	Location of Metastases	Other Treatment	Survival since Adrenalectomy	Objective Response	Subjective Response
E. M.	53	Menopause with androgens 3½ yr. prior	†	Contralateral breast	Radiation; androgens	10 mo.	Regression of chest wall lesions	Patient asymptomatic prior to adrenalectomy
E. R.	42	1½ yr. postmenopausal (radiation)	†	Osseous, lymph nodes	Radiation; androgens	Died	None	None
L. B.	39	3 yr. postmenopausal (surgical)	7 yr.	Osseous	Radiation; estrogens; androgens	7 mo.	None	Cessation of pain; ambulatory again
I. B.	43	Premenopausal	2 yr., 2 mo.	Soft tissue, pleural effusion	Radiation; androgens	Postoperative death	.....	.....
F. B.	51	6 yr. postmenopausal	5½ yr.	Pulmonary lymph nodes, liver	Radiation; androgens	4 mo. (died)	Increase in pleural lesions	None
L. C.	63	12 yr. postmenopausal	6 yr.	Osseous, pulmonary	Radiation; estrogens; androgens	6 mo. (died)	Progression of lesions	None
R. H.	40	6 mo. postmenopausal	4 yr.	Lymph nodes, liver, skin	Radiation; androgens	10 mo.	Diminution in size of liver and axillary nodes	Patient asymptomatic prior to adrenalectomy
M. K.	49	Premenopausal	5 yr.	Osseous, ovaries, adrenals, colon, peritoneum	Radiation; androgens	15 mo.	Recalcification of osteolytic lesions	Cessation of pain
L. W. W.	45	7 mo. postmenopausal (radiation)	2 yr.	Osseous	Radiation	6 mo.	Recalcification of osteolytic lesions	Cessation of pain
H. S.	57	7 yr. postmenopausal	15 yr.	Osseous, liver	Radiation; androgens; estrogens	2 mo. (died)	Rapid progression of lesions	None
N. S.	43	6 yr. postmenopausal (radiation)	6 yr.	Osseous, lymph nodes, meninges, liver, pulmonary	Radiation	Postoperative death	.....	.....
M. T.	35	3 mo. postmenopausal (radiation)	2½ yr.	Skin, osseous; ovaries, adrenals	Radiation	11 mo.	Regression of lesions; disappearance of ascites	Cessation of symptoms
V. T.	55	.....	11½ mo.	Adrenals, lymph nodes, bones	Radiation	4 mo.	Recalcification of osteolytic lesions	Cessation of pain
H. K.	58	12 yr. postmenopausal (radiation)	12 yr.	Osseous, lymph nodes	Radiation; ablation of ovaries; androgens	10 mo.	Regression of scalp masses	Cessation of pain
B. P.	46	13 yr. postmenopausal (surgical)	2 yr., 8 mo.	Osseous, skin	Radiation	9 mo.	None	Relief of pain
B. K.	57	6 yr. postmenopausal	4 yr.	Osseous	Radiation; androgens	14 mo.	None	Questionable relief of pain
M. S.	43	Menopause caused by radiation treatment to spine 2 yr. prior	2 yr.	Lymph nodes, osseous, pleural	Radiation; androgens	7 mo.	Progression of osteolytic lesions	Relief of pain for 5 mo.
B. F.	69	28 yr. postmenopausal	1½ yr.	Pulmonary lymph nodes, soft tissue	Radiation	9 mo.	Progression of metastases	None
M. W.	48	10 yr. postmenopausal	12 yr.	Pulmonary, osseous	Estrogens; androgens	34 mo.	Progression of lesions	Cessation of pain for 19 mo.
M. O.	57	Postmenopausal	†	Pulmonary, skin	Radiation	1 yr.	Epithelialization of ulceration	Disappearance of hemoptysis and cough
J. D.	60	10 yr. postmenopausal	10 yr.	Pulmonary pleura, lymph nodes, rib, mass at base of sternum	.....	1½ mo.	Cessation of pleural effusion	Cessation of pain

* Five cases which are too recent for proper evaluation are not tabulated.
† Patient had not had mastectomy.

2 or 3 days, you should increase by 1 the number of tablets you have been taking.

"If this simple change in the amount of salt taken does not stop the weight change then you should see your doctor.

"Since you are a little bit unusual without your adrenals you should always carry the note concerning your operation with you so that in the event of your being knocked unconscious in an accident the doctors taking care of you would know that you have no adrenals.

"Life without adrenals is perfectly all right except that some people get a dark pigmentation. There is not too much we can do about it but at worst you look as if you had a vacation in the desert when everyone else is pale."

Patients should be seen at frequent intervals. They should keep a record of their daily weight, and should be taught to increase the sodium chloride intake with weight loss and to reduce it with weight gain and anasarca. Blood pressure, both recumbent and supine, should be determined at each visit so as to detect the presence of postural hypotension which requires additional desoxycorticosterone. The patient should be instructed on the necessity of increasing the intake of cortisone during intercurrent disease, poor food intake or surgical emergencies. She should carry a card stating she has been adrenalectomized and giving her basal replacement therapy, with a warning to notify the attending surgeon in case of emergency. This will prevent post-traumatic death due to adrenal insufficiency.

Results. Fifty patients have undergone bilateral adrenalectomy and oophorectomy during the past fifty-one months. (Tables III and IV.)

Two patients died postoperatively; both were moribund at the time of the operation. One of these was markedly jaundiced and had extensive liver metastases; the other was markedly debilitated. With one exception patients with jaundice or extensive liver metastases did poorly, and it is now apparent that the procedure is contraindicated in the presence of either.

The follow-up period varied from one to thirty-four months. Twenty-one patients expired from one to eighteen months following operation. Eighteen patients showed objective evidence of improvement. Twenty-eight patients improved subjectively and fifteen failed to show any favorable effects. The longest period of improvement was twenty-eight months.

It must be stressed that the selection of acceptable criteria for proper evaluation of objective improvement presents serious difficulties. The reduction in size of tumor mass, the healing of ulcerations, the roentgenologic evidence of recalcification of an osteolytic lesion, and the disappearance of pleural effusions and abdominal ascites were interpreted as justifiable evidence of improvement.

TABLE IV

SUMMARY OF RESULTS IN FIFTY CASES OF BILATERAL ADRENALECTOMY AND OOPHORECTOMY FOR MAMMARY CARCINOMA (NOVEMBER 1952 TO JANUARY 1955)

Result	Number	Per cent
Postoperative deaths	2	4
Expired subsequently	22	44
Still living (2 to 34 mo. after operation)	26	52
Improved objectively*	16	36
Improved subjectively*	21	47
Failed to respond*	19	42

* Five cases which are too recent for proper evaluation are not tabulated.

Subjective improvement was noted more frequently than objective change. It was most dramatic in bedridden patients with widespread osseous metastases. Cessation of pain usually occurred immediately following operation or within the first twenty-four hours. Paradoxically this also occurred in the presence of objective evidence of progression of the lesions.

The absence of estrogens and the administration of cortisone tend to produce a gradually increasing osteoporosis; pigmentation is rarely a problem unless more than 37.5 mg. of cortisone are administered daily.

SUMMARY

1. Bilateral adrenalectomy and oophorectomy have been performed in fifty patients.

2. The pre- and postoperative care as well as the operative technic employed have been described and illustrated.

3. Objective evidence of changes in the metastatic lesions were observed in sixteen patients (36 per cent). Marked subjective relief of symptoms occurred in twenty-one patients. The relief of subjective symptoms is

more pronounced and lasts longer than that obtained with medical adrenalectomy.

4. Expert management of substitution therapy of adrenalectomized patients is a most essential part of their care.

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DISCUSSION

Peter H. Forsham (San Francisco, Calif.): The results reported in this series of fifty patients with metastatic carcinoma of the breast who have had bilateral oophorectomy and adrenalectomy are gratifying. They agree very closely with those obtained by other groups, such as Dr. Huggins' in Chicago and Dr. West's in New York.

The principal aim in this procedure is the elimination of all estrogens, on the assumption that this hormone supports the growth of carcinoma of the breast. This was postulated as early as 1886 by Beatson, who reported on the beneficial effect of menopause and ovariectomy in metastatic carcinoma of the breast. Dr. T. L. Dao, working with Dr. Huggins, has published data on urinary estrogen which demonstrate that after castration alone half the amount of the previous estrogen is still present in the urine. However, after removal of both adrenals estrogens can no longer be detected. The procedure is not uniformly successful because, unfortunately, less than half of the carcinomas of the breast appear to be fully dependent on estrogen. Failures cannot be attributed to extra adrenal cortical tissue left behind at surgery, because the cortisone used in replacement therapy will, by suppressing the patient's ACTH production, prevent any stimulation of such adrenal cortical rests and their production of estrogen.

One might mention the recent success of "medical" adrenalectomy, in which bilateral ovariectomy is followed by the administration of from 50 to 200 mg. of cortisone acetate or hydrocortisone daily. It appears that the metastatic carcinoma often escapes under treatment with the small (50 mg. or less) suppressant doses and that larger amounts will again arrest the metastatic spread. One is dealing either with the effect of estrogen suppression or with a peripheral catabolic action on the metastases themselves when these large doses of cortisone are used. Whether this medical procedure will replace surgical adernalectomy in the future will be determined by further experience. Since large doses are required, the many side effects make this treatment more hazardous and cumbersome.

As one who grew up during a period when removal of both adrenals meant a fatal outcome unless large and impractical amounts of adrenal cortical extracts were used, it has been very gratifying to see how well patients do with modern substitution therapy. It might be pointed out that the scheme presented by Dr. Galante differs somewhat from that used by Dr. Huggins and his group, in that desoxycorticosterone, a pure salt retainer, is not used early in the management. This is indeed unnecessary, since with the large amounts of cortisone all the salt and water retention necessary is obtained and blood pressure is sustained. Using that form of therapy one need not be concerned with edema and the low potassium syndrome which large amounts of desoxycorticosterone are prone to induce. Within the very near future a water-soluble preparation of hydrocortisone will become available. With that preparation one need merely give 100 mg. at the time of the incision and every six hours during the first day; every eight hours during the second day, and then continue with the oral cortisone acetate for the third day as outlined by Dr. Galante. Since 50 per cent of the hydrocortisone administered intravenously or intramuscularly is eliminated from the body within four hours, there is little danger of overdosage; and

yet the patient is very adequately prepared for surgery.

The real and unsolved problem in the management of these patients is the osteoporosis which develops and persists with the necessary long-term cortisone therapy. The main factors are: (1) the absence of anabolic endogenous estrogen and androgen, (2) the catabolic effect of the cortisone.

We find that osteoporosis becomes clinically noticeable between six and nine months after this procedure, and in severe cases one may have to give androgens to minimize this complication.

The procedure as discussed has already been supplemented by an experimental one of promise at the University of California in Berkeley, namely, hypophysectomy and very recently, proton bombardment of the pituitary. Elimination of growth hormone has been shown to slow down the growth of neoplasms both in animals and in patients with panhypopituitarism, and preliminary results in various centers are sufficiently encouraging to warrant hope that our armamentarium has not yet been exhausted.

Bilateral adrenalectomy and oophorectomy should no longer be considered an experimental procedure but rather an established form of therapy in patients with metastatic carcinoma of the breast who no longer respond to further surgery, x-ray therapy and various changes in hormonal balance.

[Following are several questions asked from the floor which were answered by Dr. Galante.]

EUGENE J. JOERGENSON (Glendale, Calif.): Do you find that these patients lose much sodium after adrenalectomy, and how do you follow them in this regard?

ANSWER: We have not found it necessary to measure quantitatively the amount of sodium excreted in the urine after adrenalectomy. An accurate daily check of the patient's weight has been found to be a good indication of any fluctuation in sodium excretion. The patient is given detailed instructions as to the amounts of sodium they must take. Sodium chloride intake is increased by 1 to 5 gm. in addition to the dietary intake if the patient loses weight. An increase in body weight is an indication to decrease the amount of sodium chloride taken. We have had no difficulty with these forty-six patients in regard to sodium retention.

LESTER R. CHAUNCEY (Portland, Ore.): Have any of the patients who have not responded to adrenalectomy been subjected to re-exploration in order to rule out the presence of accessory adrenal tissue? What is the value of performing oophorectomy in a patient who is sixty years old and past her menopause?

ANSWER: We did not re-explore any patients to find out whether they had accessory adrenal tissue, for the reason that the amount of cortisone given for replacement therapy suppresses any accessory tissue that might be present at the time. The value of performing surgical oophorectomy in patients who are old and past their menopause consists of the fact that it is possible to detect measurable amounts of estrogens in the urine of these patients. This estrogen is in part produced by the ovaries because it decreases markedly after oophorectomy. By removing the adrenal glands one attempts to remove one of the remaining major sources of estrogens.

HOWARD C. STEARNS (Portland, Ore.): Is it worth while for the patient to undergo this procedure?

ANSWER: Obviously with regard to the patients who show a response to the procedure I think it is worth while, from the point of view of both the patient and the physician. Some patients have had anywhere from six months to two years of fairly comfortable existence and a few patients have been able to return to their gainful occupations. The unfortunate feature is that we cannot predict which patient will respond, and if so, the duration of the response. One patient is fifty-four months postadrenalectomy at the present time. She is undergoing radiation to her pituitary gland because of a relapse. Pituitary ablation by means of irradiation is a procedure that is currently under study by other investigators.

Cardiac Arrest—Problems in Its Control

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IN the past most unexpected fatalities occurring in the operating room have been diagnosed as anesthetic deaths. In recent years there has been an increasing awareness that in many such instances "death" followed ventricular asystole.[1–5] Further, prompt recognition of such a situation has frequently provided the basis for correction by appropriate resuscitative treatment.[6–9] Certain problems involved in the control of this complication have been studied by us, and our present opinions regarding them will be presented.

Not infrequently in the operating room an erroneous diagnosis of death is made when only ventricular asystole is present. This mistake is dependent upon failure to recognize that under such circumstances death involves not only the existence of ventricular asystole, but also the continuation of this condition for a period of time. The time factor is that period of minutes required for progressive hypoxia to produce lethal systemic damage. A further error may develop when, under those conditions, the ensuing death is ascribed to the primary or some complicating disease. The frequency of this latter error will influence the percentage of operating room deaths excluded from the incidence of "cardiac arrest" reported from any institution. This may be very frequent because in the opinion of many physicians cardiac arrest is a condition occurring as an unexpected catastrophe in good risk patients. Actually the factors predisposing to cardiac arrest occur much more frequently and to a greater degree in seriously ill patients.

Because of the foregoing considerations consistent analysis of such problems requires a definition of cardiac arrest. An analysis of our material at the Los Angeles County Hospital has led us to develop the following definition for the purposes of this report: Cardiac arrest is potentially avoidable, usually reversible ventricular asystole, with or without fibrillation, precipitated by the effects of anesthesia alone, of surgery alone, or of these two in combination.

Excluded from this definition are those instances of fatal ventricular asystole occurring in the operating room in which the disease process was so severe that the physiopathologic mechanisms were irreversible or when uncontrollable conditions such as fatal myocardial or pulmonary infarction occurred. Evidence will be presented to justify the criteria in this definition.

We should like to emphasize that cases of "cardiac arrest" are not due to some mysterious entity such as "status thymolymphaticus," but rather that a diligent search will almost always reveal etiologic factors conditioning the development of this catastrophe. This, of course, necessitates an impartial objective critique.

In the investigation of the individual case it must be borne in mind that the anesthetic agents employed are all potentially lethal, as are many of the complicated surgical technics necessary to permit alleviation of susceptible disease processes. Under these circumstances a narrow margin of safety often prevails so that even a small human error in judgment may produce a fatal result. The surgical procedure itself may be capable of producing physiologic disturbances, such as hypoxia, which are in essence the same as those that may result from the anesthesia. An example of such hypoxia is that produced by massive operative hemorrhage. If the incidence of these catastrophes is to be reduced, it is vitally important that the patient be so prepared for operation that he is in the optimum condition to withstand the proposed procedure. This applies especially to such problems as hypovolemia, anemia, hypermetabolism, and fluid and electrolyte imbalances. If these conditions are not adequately treated preoperatively, the margin of safety is

unnecessarily reduced. If, then, an unduly prolonged or complicated operative procedure is superimposed, "cardiac arrest" may ensue. A feeling of mutual responsibility between the surgeon and the anesthesiologist should exist to see that optimum conditions for the patient's safety prevail at the time he arrives in the operating room. Good judgment and careful management should continue throughout the anesthetic and surgical procedure. This will greatly reduce the incidence of "cardiac arrest." Such mutual responsibility in one of its simplest but most effective phases is exemplified by a surgeon who immediately reports the presence of abnormally dark blood in the operative field. Because the colorimeter he has to read is much more sensitive than that available to the anesthesiologist, it is unjustifiable to assume that all matters related to oxygenation of the patient are the sole responsibility of the anesthesiologist.

Etiology. We have attempted to classify the important causes of cardiac arrest. It is hoped that, as with the problem of definition, the classification will serve to correlate and clarify the etiologic factors. In developing this outline we have drawn on the many publications on cardiac arrest as well as on our own experience. This experience is largely based on eighty-two instances of cardiac arrest from the Los Angeles County Hospital during the last four years. Additionally, our experience extends to the same institution previous to 1951, and to several private hospitals. Further, this includes our observations in the laboratory where approximately sixty dogs repeatedly have been put into cardiac arrest and resuscitated.

CLASSIFICATION OF CAUSES OF CARDIAC ARREST

I. Respiratory
  A. *Asphyxia* (hypoxia plus hypercapnia)
    1. Airway obstruction
    2. Aspiration of vomitus
    3. Excessive tracheobronchial secretions
    4. Prolonged inadequate ventilation
    5. Respiratory depression
      a. Too high spinal
      b. Overdose of premedication
  B. Hypoxia (without hypercapnia)
    1. Unrecognized empty oxygen tank
    2. Improper connection of gases
    3. Mechanical difficulties with anesthesia machine
    4. Low oxygen content in anesthetic mixture
  C. Hypercapnia (without hypoxia)
    1. Inadequate ventilation with high oxygen content
    2. Ineffective or absent carbon dioxide absorber
    3. Rapid elimination of hypercapnia (posthypercapnic syndrome: "washing out" of CO_2)
II. Cardiovascular-hematologic
  A. Decreased cardiac output
    1. Cardiac disease
    2. Inadequate right heart filling
      a. Increased peripheral vascular bed
        (1) Sudden change of position
        (2) Spinal or epidural anesthesia
        (3) Vasodilating drugs
      b. Improperly applied positive pressure ventilation
        (1) Period allowed for expiration too short
        (2) Maintenance of too much positive pressure during expiratory phase of cycle
      c. Mechanical interference
        (1) Severe break in operating table
        (2) Operative compression of vena cava
    3. Hypovolemia (shock)
      a. *Inadequate preoperative correction*
      b. *Operative hemorrhage*
  B. Anemia
III. Metabolic
  A. Hyperpyrexia
  B. Electrolyte imbalance
  C. Hyperazotemia
  D. Prolonged, exhausting stress
  E. Malnutrition
IV. Pharmacologic
  A. Myocardial depression
    1. *Relative overdose of anesthetic agent*
    2. Overdose of preanesthetic medication

B. Drug incompatibility
C. Drug hypersensitivity

v. Neurologic-psychologic
A. Vagal effect
1. Tracheal or esophageal trauma
2. Direct vagus nerve trauma
3. Mesenteric or visceral traction
4. Inadequate preparation with atropine
B. Anxiety

The italicized items represent the commonly encountered causes recognized in our series. We did not encounter any cases that could be classified in the neurologic-psychologic group, but so much discussion has been directed toward the factors of anxiety and vagal influence that their inclusion was considered necessary.

ANALYSIS OF CASES

At the Los Angeles County Hospital, during the four years 1951 to 1954 inclusive, there were 102 instances of ventricular asystole occurring in conjunction with anesthesia and surgery. This statistical information is presented in Table I. Examination of this material with reference to our definition of "cardiac arrest" determined that twenty of these cases should not be considered to have been instances of cardiac arrest. Death in these patients, we believe, was due to the severity of the disease and was unrelated to anesthesia and surgery. The evidence for this conclusion is shown in Table II. Each case included in this group presented a problem which seemed by itself to have been lethal. These patients were all anesthetized. In all probability the initial mechanism precipitating death was ventricular asystole. However, the coexistant systemic lesions were so severe that resuscitation was impossible. Since the reversibility potential did not exist, they are considered as deaths rather than as cardiac arrests.

There were difficult decisions regarding cases to be listed in Table II. An illustrative case may be cited. A sixty-nine year old woman was admitted with a duodenal ulcer perforation of thirty-six hours' duration. In spite of intravenous fluids and transfusion no blood pressure could be obtained until an intravenous neosynephrine® drip was started. A blood pressure reading of 90 systolic and 60 diastolic was then obtained. The abdomen was opened under endotracheal cyclopropane anesthesia. As the peritoneum was incised, cardiac arrest occurred. Thoracotomy was performed in less than one minute and resuscitation followed twenty seconds of cardiac massage. Closure of the duodenal perforation was then accomplished and the patient made a complete recovery. Had the cardiac massage not been done, this case (not placed in the group in Table II) could easily have been classified as death due to severe shock and placed in this group. This instance of cardiac arrest further illustrates the danger of a concept of cardiac arrest restricted to good risk patients. Actually, patients such as the one just cited enter the operating room greatly predisposed to this complication.

TABLE I
OPERATING ROOM VENTRICULAR ASYSTOLES
(LOS ANGELES COUNTY HOSPITAL)

Total ventricular asystoles	102
Death attributable to disease	20
"Cardiac arrest"	82
With fatal outcome	65
With complete recovery	17
Total operations (1951 to 1954 inclusive): approx. 48,000	
Incidence of "cardiac arrest": approx. 1:585	

TABLE II
ETIOLOGIC FACTOR IN TWENTY CASES OF OPERATING ROOM DEATHS ATTRIBUTABLE TO DISEASE

Cerebral contusion	5
Pulmonary embolism	3
Hemorrhage, uncontrollable, non-operative	3
Cardiac tamponade	2
Brain tumor, terminal	2
Uncorrectable shock	2
Myocardial infarction	1
Pneumothorax and atelectasis	1
Unknown*	1

*No necropsy; evidence indicated myocardial infarction.

After exclusion of these twenty cases of death attributable to disease, eighty-two remained that are classified as "cardiac arrest." Since in this four-year period there have been approximately 48,000 operations, this yields a cardiac arrest incidence of approximately 1 in 585 operations. This high incidence has served to stimulate our study. Since many of these patients had severe disease and duration effects, the margin of safety with which the resident anesthetist and surgeon had to deal was small. Perhaps in some of them this margin was so small that they might have been classified more appropriately as death attributable to disease. Each of the eighty-two cases has been

carefully analyzed from a detailed record. Complete records were kept during surgery and a detailed analysis was prepared by the anesthesiologist. Information from the other members of the operating team was included in this report. A critique was then written by the Director of Anesthesia. This was completed within twenty-four hours after the arrest occurred. This analysis has shown that in every instance one or more detectable etiologic mechanisms for cardiac arrest existed.

TABLE III
TYPE OF OPERATION IN EIGHTY-TWO CASES OF "CARDIAC ARREST"

Abdominal	40
Chest	16
Cardiac	2
Other than cardiac	14
Orthopedic	10
Hip fracture	5
Other fractures	2
Amputation	3
Neurosurgical	5
Urologic	3
Nephrectomy	2
Prostatectomy	1
Repair skin lacerations	3
Others	5
Thyroidectomy	1
Tonsillectomy	1
Repair cleft palate	1
Rectal fissurectomy	1
Radical mastectomy	1

TABLE IV
RESULTS OF MANAGEMENT IN EIGHTY-TWO CASES OF "CARDIAC ARREST"

Cardiac resuscitation not attempted	25
Cardiac resuscitation attempted	57
Without success	19
Heart successfully resuscitated for a period up to 24 hr	11
Heart successfully resuscitated for a period greater than 24 hr. but with eventual death	10
Complete recovery	17

Table III categorizes the type of operation undertaken. Approximately one-half of these were abdominal procedures. Of special interest is the occurrence of only two "cardiac arrests" among approximately 200 patients undergoing cardiac surgery. Perhaps the explanation here lies in the apprehension that the anesthesiologist and surgeon have for these cases. They are managed very carefully. A medical cardiologist is part of the operating team, and a continuous electrocardiogram is taken. Under these conditions an electrocardiogram is of very great value in detecting relatively minimal alterations in cardiac activity of which neither anesthesiologist or surgeon may be aware. This relatively low incidence in these poor risk patients lends support to the hypothesis that "cardiac arrest" is preventable and that better management will reduce the over-all incidence.

TABLE V
PRINCIPAL ETIOLOGIC FACTOR IN SIXTY-FIVE CASES OF "CARDIAC ARREST" WITH FATAL OUTCOME

Asphyxia	28
Relative overdose of anesthetic agent	10
Operative hemorrhage	10
Inadequate preoperative preparation	9
Hypoxia	3
Vascular obstruction	1
Unknown	4

TABLE VI
PRINCIPAL ETIOLOGIC FACTOR IN SEVENTEEN CASES OF "CARDIAC ARREST" WITH COMPLETE RECOVERY

Relative overdose of anesthetic agent	8
Asphyxia	8
Unknown	1

Table IV summarizes the results of management of the eighty-two cases of "cardiac arrest." Resuscitation was not attempted in twenty-five. Of the fifty-seven cases in which resuscitation was attempted, seventeen made a complete recovery. The heart itself was resuscitated in another twenty-one cases, but death eventually occurred. Most of these latter patients remained in coma until death. The longest survival was three months.

In the sixty-five cases of "cardiac arrest" which terminated fatally, it was frequently difficult to designate a single etiologic mechanism. Table V lists the dominant factor that precipitated the catastrophe. Asphyxia was the factor most often encountered. In the nine cases listed as inadequate preoperative preparation the arrest occurred either from the effects of anesthesia or stress of prolonged surgery. However, these patients were taken to the operating room in a state of preparation that could have been improved. Their margin of safety was thereby severely jeopardized. We believe that adequate preparation of these patients might well have averted the cardiac arrest and death.

Whereas in several instances it was difficult to identify the dominant etiologic factor in those patients who had fatal "cardiac arrest," it was very clear cut in those who survived. Table VI shows that all of these, with one exception, were produced by a relative overdose of the anesthetic agent or by asphyxia. In the patient designated as "unknown," no definite

cause could be ascribed. "Cardiac arrest" suddenly occurred in this seventy-seven year old patient while the incision was being closed two and a half hours after start of left hemicolectomy. A spinal anesthetic, with no supplement, was employed. Complete recovery followed transabdominal cardiac massage.

TABLE VII
VENTRICULAR ASYSTOLE IN FIFTY-TWO CASES OF "CARDIAC ARREST"

Type	Died	Recovered
Ventricular standstill (45).....	29	16
Ventricular fibrillation (7).....	6	1

Information on the type of ventricular asystole was available in fifty-two of the cases, as indicated in Table VII. Ventricular fibrillation accounted for seven of these, one of which was successfully resuscitated.

PROGRAM FOR PREVENTION AND TREATMENT OF CARDIAC ARREST

The Los Angeles County Heart Association has been exceedingly active in its effort to combat the problem of cardiac arrest. A cardiac resuscitation committee of the Association has been functioning for three years. Its primary function has been to provide information on this subject to all practitioners in the area. Nearly all hospitals have had at least one monthly staff meeting devoted to this problem. These meetings were conducted by one group of four or five physicians representing the Heart Association who visited each hospital in turn. Further, on each hospital staff one or two surgeons were designated to coordinate the hospital activities in this regard. They were held responsible for setting up the "cardiac resuscitation kit" in such a manner that it was complete, readily available and conformed with the criteria established by the Los Angeles County Heart Association. Formal lectures were held for the responsible surgical nurses regarding the accepted plan of attack in the event of this emergency so that they could better assist the operating room physicians. It was the designated surgeons' further responsibility to see that the special placards, entitled "Program of Action—Cardiac Arrest," were permanently and conspicuously placed in each scrub room and operating room so that all operating surgeons would have occasion to review the significant steps frequently and thus become more familiar with them.

In addition, the Los Angeles County Heart Association and the University of Southern California School of Medicine established a practical course in cardiac resuscitation using dogs. This is a one-session, two-hour course, available to all resident physicians as well as all practitioners in the area. It is conducted one evening each week and has now been in operation for eight months. It has been so enthusiastically received, even by those well informed on the subject, that we believe it could be adopted to advantage in other areas. Experience has taught us that, valuable as they are, many hours spent in reading and attending lectures on cardiac arrest do not compare in effectiveness with this one evening period of practical training.

The course is conducted by a surgeon and an anesthesiologist. Experience has shown that each session should be limited to four students. An initial period of thirty to forty minutes is spent in lecture during which the practical aspects of cardiac resuscitation are presented. No effort is made to discuss many of the hypothetic aspects of the problem. The following points are stressed: Etiologic factors for and recognition of arrest with danger signs of impending arrest; duty of the anesthesiologist; opening the chest; effective massage of the heart; drugs of value; necessary steps required to re-establish spontaneous heart action; closure of the chest; postoperative features. The remainder of the time is spent with anesthetized dogs in the application of the principles discussed. Ventricular asystole is produced by acute hypoxia in one dog and acute asphyxia in another. Hypoxia is produced by positive pressure ventilation with 100 per cent nitrous oxide with soda lime absorption. The resultant ventricular asystole occurs in about eight minutes and is a ventricular standstill. Care must be used to accomplish ventilation good enough to remove all excess carbon dioxide. If carbon dioxide is allowed to accumulate, the rapid elimination of this hypercapnia during oxygen ventilation in treatment of the standstill will result in conversion of the standstill to fibrillation. During this time the electrocardiographic features are demonstrated. These characteristically involve an initial bradycardia followed by tachycardia and then a severe

terminal bradycardia. Persistent high amplitude QRS complexes continue long after visible ventricular contractions have ceased. This phenomenon is not common knowledge. Many physicians believe that with the onset of cardiac arrest the electrocardiographic tracing abruptly becomes flat. This error was responsible for the failure to recognize arrest promptly in one of our clinical cases. The tracing showed continued cardiac activity for twenty minutes after neither pulse nor blood pressure was obtainable.

The chest is opened by the instructor. No effort toward asepsis is observed. The significant anatomic details are pointed out and the pericardium incised. The anesthesiologist delivers 100 per cent oxygen and the instructor demonstrates cardiac massage. The students check the effectiveness of this massage by palpation of the femoral arteries. The cardiac standstill is resuscitated. Ventricular fibrillation is then produced by low amperage electric shock. Massage is reinstituted with the heart in fibrillation, and each student practices the technic of massage and his effectiveness is checked by palpation of the femoral arteries. At first the majority of the students are unable to produce an effective massage. The instructor frequently finds it necessary to spend considerable time with the student before the cardiac massage produces a good femoral pulse. We have been impressed with this high incidence of ineffective massage when it is done by physicians for the first time. Perhaps this factor is significantly responsible for the high incidence of unsuccessful resuscitations reported in the literature. The fibrillation is then converted by higher amperage electric shock. Each student then fibrillates, massages and defibrillates until the heart can no longer be resuscitated.

Ventricular asystole is then produced in a second dog by asphyxia. This is accomplished by clamping off the endotracheal tube, producing hypercapnia and hypoxia. Approximately twelve minutes are required to produce ventricular standstill. The ventricular standstill thus produced usually is converted to ventricular fibrillation after approximately one to two minutes of positive pressure oxygen ventilation given in association with cardiac massage. This conversion to fibrillation appears to be due to rapid elimination of hypercapnia.[10–19] This emphasizes to the student another mechanism of cardiac arrest. Each student again practices cardiac massage and defibrillation. Drug therapy in each of the animals is carried out in accordance with the principles to be presented in the following paragraphs.

TREATMENT OF CARDIAC ARREST

Recent publications are quite consistent in their recommendations for the treatment of cardiac arrest and vary only in smaller details, especially in regard to type and frequency of drug injections. We advise the following program.

When cardiac arrest is suspected by the anesthesiologist's inability to palpate a peripheral pulse or obtain a systolic blood pressure, the surgeon palpates the femoral vessels. If an abdominal incision has been made, the aorta or the heart is palpated through the diaphragm. If a palpable pulse is not obtained, the diagnosis of cardiac arrest is confirmed.

We caution against using the stethoscope to listen for heart sounds, waiting for an electrocardiogram, or readjusting the sphygmomanometer cuff. These maneuvers are of little value and waste time when even seconds are important. Although experimentally the cerebrum can tolerate cessation of oxygenation for three to four minutes, the situation is not always comparable clinically. The students are instructed that they are not to consider they have this margin of time, but rather that this period probably has nearly elapsed by the time cardiac arrest has been recognized. The surgeon calls for the cardiac resuscitation kit. The anesthesiologist ventilates the lungs with 100 per cent oxygen. An endotracheal tube, if not already in position, is inserted as soon as it is practical. The patient is placed in the Trendelenburg position. Mouth-to-mouth breathing can be maintained for a short time if an anesthesiologist is not immediately available.

If the abdomen has been opened, the surgeon repeatedly compresses the heart against the posterior aspect of the sternum through the intact diaphragm. This maneuver alone accounted for five of the seventeen complete recoveries reported in this series. If ineffective, cardiac massage done in this manner should be continued for no longer than ten seconds. If effective, the resuscitation has probably been due to mechanical myocardial stimulation. Effective circulation can be produced only by

manual compression of the heart through the open chest. Direct visualization and palpation of the heart is required for satisfactory drug injections. This is necessary to insure the proper site of injection as well as to avoid injury to the coronary vessels. We do not recommend incision of the diaphragm.

The surgeon opens the left chest through an incision extending from approximately the lateral sternal border to the mid-axillary line below the left breast. Usual asepsis is not followed. No effort is made to count the interspaces. After the intercostal incision has been made, it is helpful if the costal cartilages above and below the incised interspace are cut across adjacent to the sternum. This allows for easier introduction of the hand into the chest. A rib spreader will improve the exposure. The heart is then compressed against the posterior aspect of the sternum for five or six vigorous thrusts. If this maneuver is ineffective in restoring spontaneous heart action, the pericardium is opened widely, longitudinally, anterior to the phrenic nerve. We do not believe that effective cardiac massage can be accomplished through the intact pericardium.

Cardiac massage is then instituted with one or both hands, using sufficiently forceful compression so that an assistant is able to palpate a peripheral pulse. If a peripheral pulse cannot be palpated, the character of the cardiac massage is considered unsatisfactory. Once effective massage is attained it is continued for two to three minutes in order to reoxygenate the cerebrum and myocardium. The rate of cardiac compression should be 60 to 100 per minute. If spontaneous heart action does not take place by this procedure alone, drug treatment is required. Epinephrine, calcium chloride and procaine solutions are kept ready for immediate use in vials of 50 cc. in the cardiac resuscitation kit. Each is so prepared that the therapeutic dose is 10 cc. Thus the surgeon, regardless of the drug which he wishes to use, needs to remember only that 10 cc. of the prepared solution contains the correct amount of drug and the bottle is so labeled. The program to be instituted from this point will depend upon whether the cardiac arrest is due to ventricular standstill or ventricular fibrillation. If due to ventricular standstill, which comprises about 85 to 90 per cent of these catastrophes, the following program is instituted:

For ventricular standstill 0.3 cc. of 1:1000 dilution of epinephrine (10 cc. of the prepared solution) is injected directly into the chamber of the left or right ventricle. Cardiac massage is interrupted for only as long as it is required to complete the injection. Massage is then resumed; and if after three or four minutes only feeble ventricular contractions occur, 3 cc. of 10 per cent solution of calcium chloride (diluted to 10 cc.) is injected and massage is continued. As long as the ventricular contractions are by themselves ineffectual, the surgeon will find no difficulty in massaging the heart. As soon as the ventricular contractions become forceful he will find it impossible to continue to assist the heart, and thus easily recognize that further massage is not required.

If there is no ventricular response to the first injection of epinephrine, a second dose is given. If there is still no ventricular response, we administer calcium chloride. Massage is continued throughout. If there is no response after two injections of calcium chloride, it is probably futile to continue efforts of resuscitation. However, if but a feeble ventricular response occurs, efforts at resuscitation must be continued indefinitely. At the present time there are no other drugs that we recommend, although isopropyl nor-epinephrine, potassium chloride and atropine have been advocated.

For ventricular fibrillation, after the initial period of cardiac massage of approximately three minutes, defibrillation is attempted by momentary electric shock, using a current of about two and one-half amperes. The electrodes are placed so that as much myocardium as possible is encompassed between them. If the defibrillation is successful, ventricular standstill will be produced. The period of standstill may be temporary or prolonged. If prolonged, it must be treated in the same manner as described for standstill with the exception that epinephrine is used only as a last resort because of its potent fibrillatory effect. If the initial electric shock was ineffective, a second electric shock is applied. If this as well is ineffective, a series of five or six shocks is given in rapid succession. If this is still ineffective, 10 cc. of 1 per cent procaine is injected into the chamber of the left or right ventricle. Massage is continued for a minute or so and the electric shock is repeated. Our experience in the dog laboratory has demonstrated the effectiveness of procaine in aiding defibrillation. The resulting standstill, however, is sometimes very dif-

ficult to resuscitate due to the marked myocardial depressant action of the procaine. Consequently, we advise efforts at defibrillation with electric shock alone before using procaine.

After successful resuscitation the pericardium is partially closed. The pleural space is cleansed with saline solution, topical antibiotics are instilled, intercostal tube drainage is established and the chest is closed in routine fashion. The steps of this procedure for cardiac resuscitation are repeated in the following outline:

PROGRAM OF ACTION—CARDIAC RESUSCITATION

Confirmation of suspicion of "cardiac arrest":
1. Palpation of large artery only (aorta, or heart through diaphragm if abdomen open)

Do not:
1. Listen for heart sounds with stethoscope
2. Rely on electrocardiogram
3. Reposition blood pressure cuff

Duty of the anesthesiologist:
1. Ventilate lungs with 100 per cent oxygen
2. Intubate if not already done
3. Place patient in head-down position (5 to 10 degrees)
4. Supervise administration of blood or other fluids if indicated

Duty of surgeon:

I. Cardiac massage:
1. If abdomen open, compress heart against sternum through intact diaphragm, ten seconds maximum. If ineffective or abdomen not open:
2. Open left chest, fourth or fifth interspace, incise costal cartilages, ignore asepsis
3. Compress heart against sternum five or six times; if ineffective:
4. Open pericardium; differentiate ventricular standstill from ventricular fibrillation
5. Institute effective manual cardiac compression three to four minutes

II. Treatment of ventricular standstill:
1. Epinephrine, ventricular chamber
   (a) If no effect, second injection epinephrine (if no effect, calcium chloride, ventricular chamber); if still no effect, second injection calcium chloride
   (b) If partially effective, calcium chloride (if no improvement, second injection epinephrine); if still no improvement, second injection calcium chloride
2. Cardiac massage continued for two to three minutes between injections

III. Treatment of ventricular fibrillation:
1. Electroshock
   (a) If no effect, repeat electroshock with increased amperage (if no effect, rapid serial electroshocks); if still no effect, procaine, left ventricular chamber; repeat electroshock as mentioned before
   (b) If effective, treat ventricular standstill, if persistent, by massage and calcium chloride; use epinephrine only as last resort

SUMMARY

1. Anesthetic death usually represents unrecognized and untreated cardiac arrest occurring in a good risk patient.

2. Uncorrected physiologic disturbances in severely ill patients are often similar to those resulting from imperfect anesthesia or unduly traumatic, bloody surgery.

3. Cardiac arrest is more likely to occur in severely ill than relatively well patients.

4. Operating room deaths occurring in severely ill patients often are attributed to the primary disease. All such deaths should be included and analyzed when the incidence of cardiac arrest is reported from any series of surgical operations.

5. In our cases of cardiac arrest the etiologic factors in order of importance were: asphyxia, relative overdose of anesthetic, operative hemorrhage, inadequate preoperative preparation and hypoxia.

6. When the predominant etiologic factor was anesthetic rather than surgical, resuscitative measures were much more effective.

7. Control of the problem of cardiac arrest is basically a problem in education. The joint program of the Los Angeles County Heart Association and the University of Southern California School of Medicine is described.

8. Proficiency in the treatment of cardiac arrest can be attained best by practical experience in the laboratory.

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DISCUSSION

W. H. Snyder, Jr. (North Hollywood, Calif.): In commenting on this paper I would like to mention a study of this same subject that we prepared at the Los Angeles Childrens Hospital in 1952. We collected all general surgical cases operated upon at the Los Angeles Childrens Hospital during twenty years, including all subspecialties except thoracic surgery, so the increase was not due to cardiac or lung operations as these are not included. We divided these cases into four five-year periods. Thus, there were 14,000 operations in the first five years; 13,000 in the second, and so on. We also tabulated the number of deaths on the table for each five-year period and it ran along four, two, three per five-year period until 1947 to 1952, in which there were fourteen cardiac arrests with almost an equal number of cases operated upon. The impressive series analyzed by Drs. Mikkelsen, Denson and Berne is a further indication that surgeons and anesthetists alike are rightly concerned over this major problem.

Their paper also emphasizes the important controllable pathophysiologic factors contributing to cardiac arrest and helps the profession realize that lack of oxygen, accumulation of CO_2, excessive amounts of drugs, hemorrhage and low blood volume can account for the arrest in three-fourths of the cases. This is important as it will make us all strive harder than ever before to correct these contributing factors when possible.

I know the efforts that are being made at the Los Angeles County Hospital and despite this the incidence of this condition is not abating. Therefore, in a practical sense, I would prefer not to label the cases as errors or faults in management, or to consider every case of cardiac arrest as avoidable. To me this is far too strict a condemnation of the operating room team. It is one thing to analyze a death in retrospect and an entirely different thing to assign the blame for it to either the anesthetists or the surgeon. Perhaps there would be more wisdom in assigning the blame to the present system, which allows the use of the new potent drugs, the pressure put on the surgeons and anesthetists to operate on desperate cases, the lack of detecting devices to predict alterations in blood gases, and electrocardiographic and electroencephalographic changes, rather than on those who must assume the responsibility for caring for a critical case.

One contribution of this paper is to point out and demonstrate experimentally that pure oxygen lack, without CO_2 accumulation results in cardiac standstill whereas asphyxia, *per se*, results in ventricular fibrillation.

Another contribution has been to outline and carry out a course in cardiac arrest which gives experience with this catastrophe before it happens. As Lahey emphasized, the hardest thing to overcome is the inertia one experiences with his first patient in the operating room. If other centers

throughout the land would institute such courses, many lives would be saved.

The authors are to be congratulated for a very worth while presentation.

W. P. Longmire, Jr. (Los Angeles, Calif.): First, I want to complement the authors on a very excellent study and presentation. Second, I think that one item we are inclined to overlook at this time, in light of a lot of information that is given us, is the value of adrenalin® in the treatment of this emergency. Certainly most of us who have had some experience with cardiac arrest while treating congenital heart disease are inclined to restrict the use of adrenalin and frequently believe that it is contraindicated, depending upon the anesthetic and the time of the ventricular asystole. However, anyone who has attempted to restart the heart in a normal dog after asystole has been produced knows that there is nothing that approaches the effectiveness of adrenalin. I venture that this point sometimes is overlooked in the operating room. I know that just within the last two months I had occasion to see asystole develop in a patient who had received spinal anesthesia for a transurethral resection. All types of procedures were being used, with the exception of adrenalin. When this was given it was possible to restart the heart very promptly. I would like to call attention to the fact that other solutions, such as potassium and particularly calcium chloride, are effective in cyanotic children, but in the patient with an otherwise normal heart nothing is as effective as adrenalin.

Clarence J. Berne (closing): There are one or two points in regard to this paper about which I would like to say a word. The first has to do with this matter of incidence. It is our opinion that in many series reported, when an institution records a certain frequency of cardiac arrest, the tendency has been to take cases of this condition occurring in the good risk patients; those people in serious condition from the disease for which they are taken to the operating room are alleged to have died as a result of their disease. This has been strongly emphasized to us by one of the patients in this series, a woman about fifty years old who was in severe shock from a ruptured ulcer of about forty hours' duration. She was given blood and other therapy aimed at controlling her hypotension. Finally, on a continuous drip of neo synephrine, her pressure was raised to about 80 systolic. She was taken to surgery; and as the incision was being made in the peritoneum, ventricular asystole occurred. It is our opinion that under most circumstances this patient would have been thought to be dead and never would have been reported as a case of cardiac arrest but rather as one of death due to shock. Fortunately, the resident went right ahead and opened her chest, successfully massaged her heart and the patient left the hospital walking. In our opinion, this whole group of patients with cardiac arrest occurring under conditions such as this last one, frequently have not been reported as such. These cases often represent instances of cardiac arrest and are so reported in this series. Therefore, we think that the incidence of cardiac arrest may be much greater than many institutions have actually reported. In reporting cardiac arrests it might be well to do it as we have done, which is to report all deaths occurring in an operating room and then give a breakdown; otherwise, the incidence is not even approximately known.

I would like to say one other word in regard to this problem of trainees in the course on cardiac resuscitation. These trainees are all doctors and most are practicing surgeons. It is very common that when a group of such students consecutively massage the heart only about half of them produce anything resembling an effective femoral pulse. We wonder if there is any relationship between this and the fact that, as reported in the literature, about 70 per cent of cardiac arrests are not resuscitated. In other words, in our experience if you take a group of doctors, open a chest and have them massage a heart, the majority of them will get no effective result whatsoever. It is only after they have been trained and trained that they can produce good circulation by their manual efforts.

Another interesting sidelight on this subject is that Dr. Mikkelsen has generally taught them the two-handed method and usually they do better with this. However, with a group that we had recently almost none of them got anywhere near a good femoral pulse with the two-handed method. Each got a very good peripheral pulse, however, when we shifted them to the one-handed method. It points up two things: first, that we do not actually know the best method for massage of a heart and second, unless a doctor has practiced himself, he might easily be doing it the wrong way.

Emile Holman (San Francisco, Calif.): I cannot let the chance go by without saying a word about the excellence of this article and the superb slides and photographs. I would also like to suggest a little attention to semantics. The expression, "massage of the heart," is misleading. If you talk about massage of the heart to medical students and general practitioners, what do they think of? They do not think of rhythmic, intermittent compression of the heart, but that is what it is: manual compression of the heart, rhythmic and intermittent in character. Let us call it what it is, so that medical students may be conditioned to that concept and not to the concept of "massage."

William P. Mikkelsen (closing): I would like to thank the discussors of this paper. In spite of the attention that cardiac arrest has received in recent years, our experience would indicate that

this problem is increasing, not decreasing. In this series of 102 cases, twenty-seven have occurred during the last six months. Dr. Snyder has suggested that attention be directed to faults inherent in the anesthetic and surgical methods. Admittedly, this is where the problem originates. It seems unlikely, however, that a significant alteration in these methods is imminent. In the meantime it is our belief that emphasis must be directed to the individual and that he be especially careful in the application of the particular technic that he uses.

Time did not permit a discussion of the actual method of cardiac resuscitation that we utilize. However, in this regard we agree completely with Dr. Longmire in regard to the efficacy of epinephrine. We have found this drug to be the single most effective adjunct in treating ventricular standstill. We caution against its use, however, in cases of ventricular fibrillation that have been converted to standstill by electric shock.

I should especially like to thank Dr. Holman for his generous remarks. He raised the interesting point of the implication of the term "massage." It has been surprising how many physicians taking this course believe that cardiac resuscitation is accomplished by simple cardiac stimulation. That this occasionally occurs is not challenged, but the great majority require forceful, rhythmic ventricular compression that satisfactorily maintains circulation. We insist that this be recognized in the course on cardiac resuscitation, but since the term "massage" has been utilized so frequently in the literature, we used it today.

Hydrodynamic Factors in the Production of Aneurysms

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DURING the past year the role of certain hydrodynamic forces in the production of aneurysms has been under investigation in the

FIG. 1. Aortogram three months after application of long stenosis to thoracic aorta.

Stanford Surgical Research Laboratory. It has already been shown that these forces can produce dilatation just distal to a stenosis in a major artery,[1,2] and it was thought that by the application of certain principles it might be possible to produce fusiform aneurysms of considerable size. Stenoses have been produced in the thoracic aorta of dogs and data have been compiled from observations by means of retrograde aortograms. Only dogs having a 30 per cent or more dilatation of the aorta have been considered as having aneurysms. By measuring the aorta above and below the aneurysms and averaging the measurements, the expected diameter of the normal aorta in the region of the aneurysm is determined. The percentage increase is then obtained by dividing the expected diameter into the difference between the expected diameter and the actual diameter of the aneurysm.

Three different methods were used in attempts to produce aneurysms. In the first group of puppies a ligature was placed around the thoracic aorta in such a manner as not to constrict the lumen but to limit the vessel to its primitive narrow diameter as the puppy grew to maturity. Thus as the dog grew larger, a gradually increasing constriction was formed. There were three dogs in this group with aneurysm; the first was operated upon on January 19, 1954, a long stenosis being made by approximating a piece of split plastic tubing to the aortic wall, covering the entire circumference. Aortograms three months postoperatively showed 37 per cent dilatation of the aorta. (Fig. 1.)

The second dog in this group was operated upon on January 20, 1954, a short stenosis being made by the application of a single ligature. Three months postoperatively the aortogram showed 70 per cent dilatation. (Fig. 2.) Shortly thereafter a second operation was performed in which the aorta was tied off completely at the site of the original constriction. Aortograms three months after the second operation showed 25 per cent dilatation, and aortograms five months postoperatively showed 20 per cent dilatation.

The last dog in the group was operated upon on January 28, 1954, and the short stenosis was made. Postoperative aortogram at three months showed 50 per cent dilatation; six months, 41 per cent dilatation; seven months, 70 per cent dilatation; and nine months, 60 per cent dilatation. (Fig. 3.) On November 4, 1954, the dog was subjected to pressure and aortogram studies following the injection of adrenalin and histamine. Figure 4*a* shows the condition of the aorta and aneurysm before the injection of any drugs, the pressures above and below

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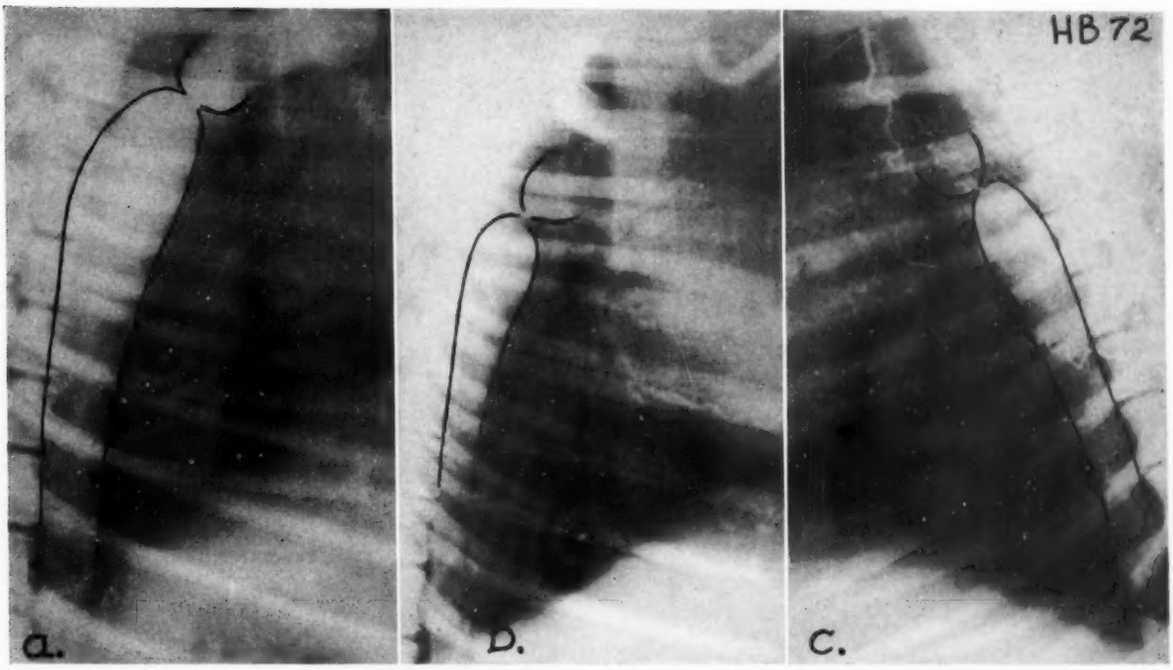

FIG. 2. Aortogram of dog after short stenosis of thoracic aorta. Aorta completely ligated at site of stenosis three months later. (*a*) 70 per cent dilatation three months after first operation; (*b*) 25 per cent dilatation three months after second operation; (*c*) 20 per cent dilatation five months after second operation.

the constriction being indicated on the aortogram. Figure 4*b* shows the condition ten seconds after the injection of 1 cc. of 1:1000 adrenalin, revealing the marked increase in size resulting from the increased blood pressure. Figure 4*c* shows the aorta fifteen seconds after the injection of 1 cc. of 1:1000 histamine with the resultant hypotension and diminution in size. Figure 4*d* was taken sixty seconds after the injection of 1 cc. of adrenalin, the aorta and aneurysm being exactly the same size as in Figure 4*b* although the pressures are somewhat less. Throughout this experiment there was little change in size of the aneurysm relative to the size of the aorta.

In the second group of animals the thoracic aorta was exposed and a partial incision was made in the wall so that the media was entered. With this incision as a starting point, a portion of the medial layer of the aorta was gently stripped off by blunt dissection. This was done over a length of 15 to 20 mm. and anywhere from two-thirds to completely around the circumference of the vessel. Figure 5 shows the thickness of the denuded area as compared with the thickness of the normal aorta. After this so-called denudation was completed, a constricting tie of umbilical tape was placed around the normal aorta just proximal to the denuded area; this tie was placed just tight enough to cause a thrill, the lumen being reduced to about one-half its normal diameter. There were four dogs in this group, all of which were two months old at the time of operation.

The first dog was operated upon on July 6, 1954. Aortograms four and six months postoperatively showed dilatations of 50 per cent and 39 per cent, respectively. (Fig. 6.) The second dog was also operated upon on July 6th, and aortograms four and six months later showed 40 per cent and 58 per cent dilatation. (Fig. 7.) The third dog was operated upon on July 13, 1954, and the aortogram four months later showed 50 per cent dilatation. The last dog in the series was also operated upon on July 13th and the aortogram three months later showed 45 per cent dilatation. (Fig. 8.)

In the last group of experimental animals, aortic denudations without constriction were performed. In these attempts little or no dilatation was produced. Two dogs with the greatest dilatation are presented as examples: The first dog had surgery on May 25, 1954, when two months old, and aortograms one-half, four and seven months later showed negligible dilatation. (Fig. 9.) The other dog had surgery on July 28th at three and a half months of age. Aortograms five months later showed practically no dilatation before and after injection of adrenalin. (Fig. 10.)

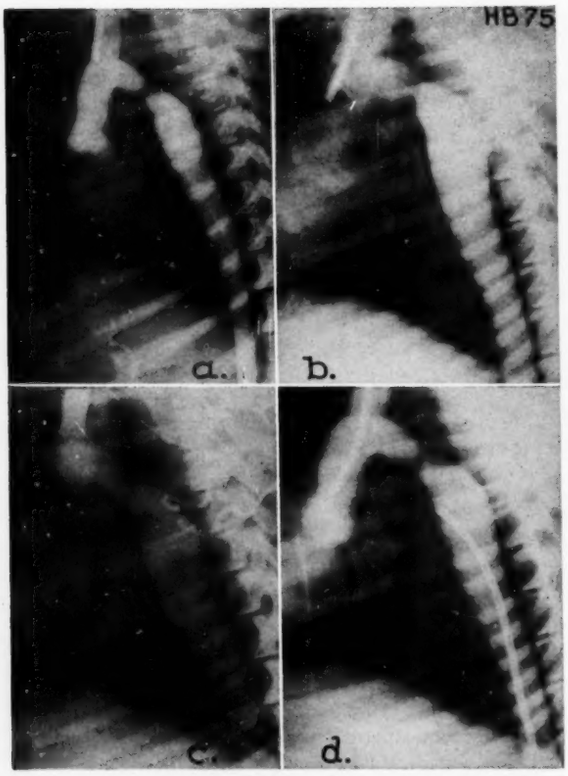

FIG. 3. Aortograms of dog after stenosis of aorta. (*a*) 50 per cent dilatation three months postoperatively; (*b*) 41 per cent dilatation six months postoperatively; (*c*) 70 per cent dilatation seven months postoperatively; (*d*) 60 per cent dilatation nine months postoperatively.

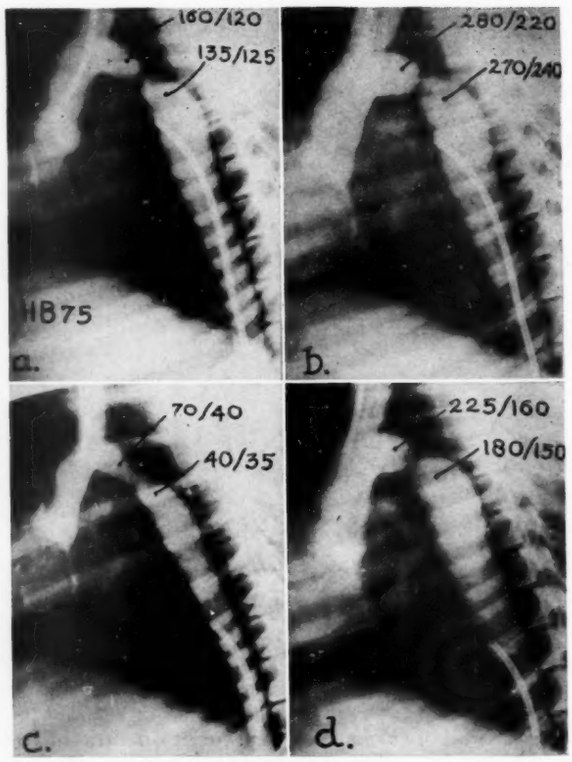

FIG. 4. Pressure and measurement studies nine months after stenosis of thoracic aorta. (*a*) Control, 60 per cent dilatation; (*b*) ten seconds after injection of 1 cc. of 1:1000 adrenalin, 42 per cent dilatation; (*c*) fifteen seconds after injection of 1 cc. of 1:1000 histamine, 38 per cent dilatation; (*d*) sixty seconds after injection of 1 cc. of 1:1000 adrenalin, 42 per cent dilatation.

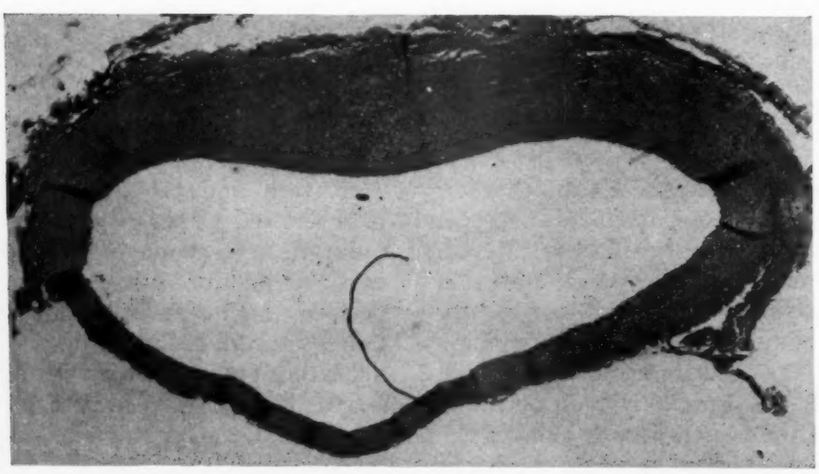

FIG. 5. Photomicrograph of denuded aorta to show relative thickness of denuded and normal wall.

Figure 11 is a photograph of the aneurysms after they were removed from two of the dogs and opened lengthwise. In number 75 the aneurysm was produced by the use of constriction only, and 60 per cent dilatation was observed; in number 105 the aneurysm was formed by constriction and denudation of the aortic wall, and 50 per cent dilatation was observed. As can be seen, there is little difference between these specimens—both have smooth intimal layers, a sharply constricted area and a thinning of the wall in the dilated portion.

Microscopic sections of the aortic wall of the first specimen above the aneurysm, at the aneurysm and below it show little of note except thinness of the wall of the aneurysm as compared with the normal wall. (Fig. 12.)

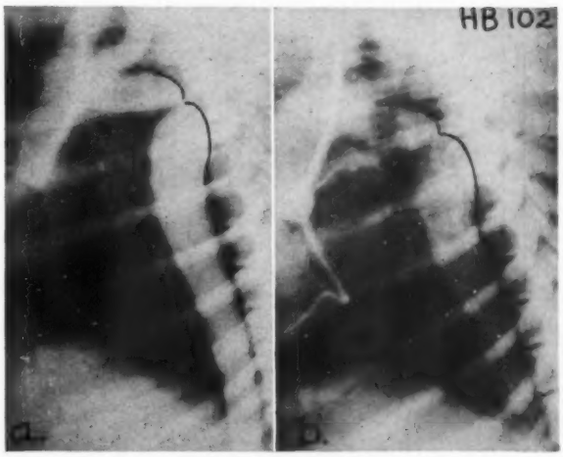

FIG. 6. Aortograms of dog after denudation and constriction of thoracic aorta. (*a*) 50 per cent dilatation four months postoperatively; (*b*) 39 per cent dilatation six months postoperatively.

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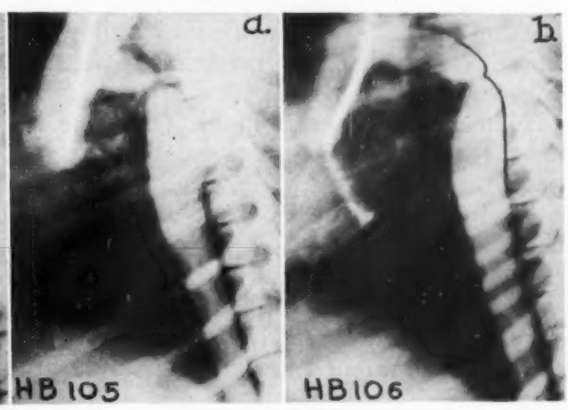

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FIG. 7. Aortograms of dog after denudation and constriction of thoracic aorta. (*a*) 40 per cent dilatation four months postoperatively; (*b*) 58 per cent dilatation six months postoperatively.

FIG. 8. Aortograms of two dogs after constriction and denudation of thoracic aorta. (*a*) 50 per cent dilatation four months postoperatively; (*b*) 45 per cent dilatation three months postoperatively.

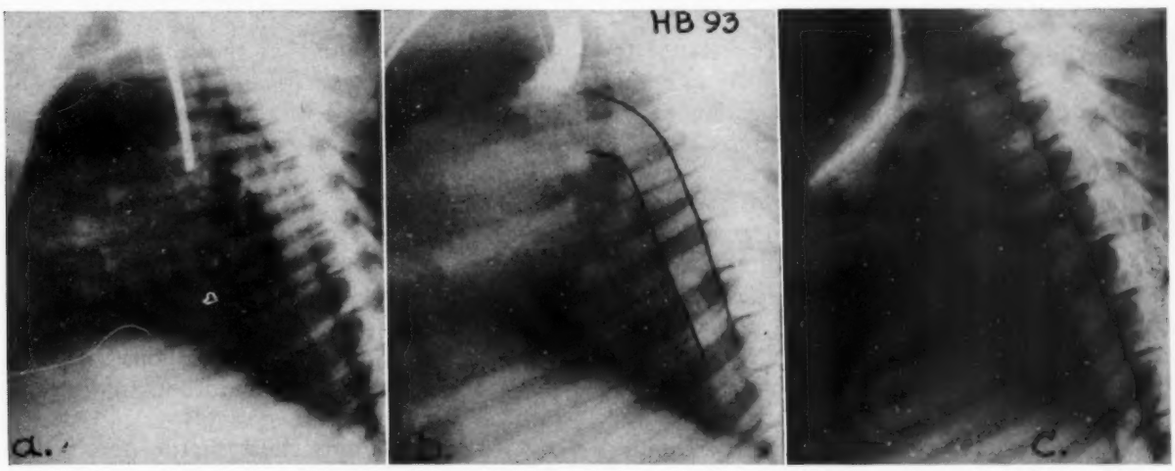

FIG. 9. Aortograms of dog after denudation only of thoracic aorta. (*a*) One-half month postoperatively; (*b*) four months postoperatively; (*c*) seven months postoperatively.

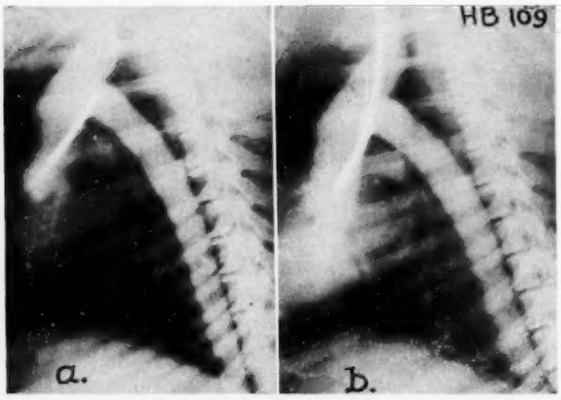

FIG. 10. Aortogram of dog five months after denudation only of thoracic aorta. (*a*) Before injection of 1 cc. of 1:1000 adrenalin; (*b*) after injection of 1 cc. of 1:1000 adrenalin.

Comparable sections from the second specimen produced by denudation in addition to constriction show that the aneurysmal wall is even thinner than in the preceding specimen. (Fig. 13.)

Hydrodynamic considerations: Of prime importance in development of the post-stenotic dilatation seen in these animals is the operation of the hydraulic laws that govern the flow of fluid through an expansile elastic tube: In the pulsating flow of blood a momentary retardation in flow occurs during diastole which is accentuated just beyond a stenosis. During systole a stream of blood emerges from the narrow stenotic channel at a greatly increased velocity as compared with the velocity in the broader channel either proximal or distal to the stenosis. Mathematically, the velocity of flow, other factors remaining the same, is inversely proportional to the square of the radius. Obviously, if a tube 18 mm. in diameter is reduced abruptly for a short distance to a diameter of 6 mm., the velocity through the constriction is increased nine times. Conversely, if a tube 6 mm. in diameter is increased abruptly to a diameter of 18 mm., the velocity will be reduced to one-ninth its previous rate. In systole, therefore, the rapidly ejected stream runs head on, as it were, into the more slowly flowing and relatively stagnant post-stenotic stream resulting in a sudden arrest in its forward flow. The high kinetic energy of the swiftly moving stream is suddenly converted into high potential energy or lateral pressure. This is the first factor responsible for the dilatation. Secondly, the clash of the two streams of differing velocities produces eddies of turbulent

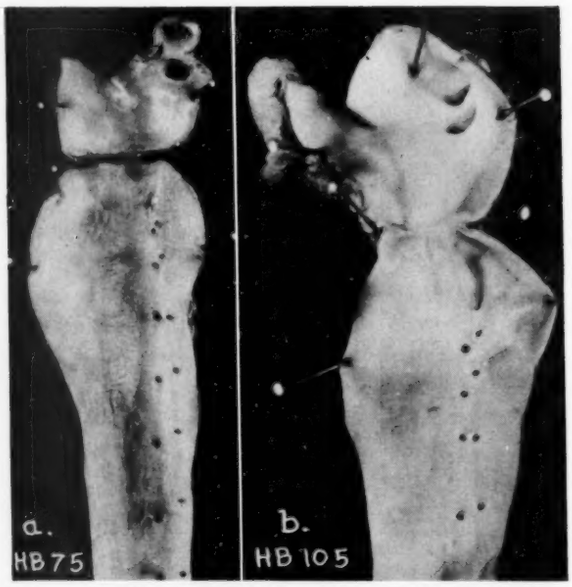

FIG. 11. Photographs of autopsy specimens after removal from dogs. There was (*a*) constriction of aorta nine months before autopsy and (*b*) constriction and denudation four months before autopsy.

and reversed flow accompanied by shocks or impacts of alternating high and low pressure which strike against the elastic wall, resulting eventually in its distention. These impacts of alternating high and low pressure also cause the vessel wall to vibrate, these vibrations being recognizable as a palpable thrill and an audible bruit.

A third factor producing dilatation beyond the stenosis is the lowered velocity due to the widening of the stream diameter resulting from the first two factors. According to the Bernoulli principle, this lowered velocity will of itself increase lateral pressure. In a pulsating stream the first two factors recur repeatedly with each systole and set the stage, as it were, for the third factor to act, also in repetitive manner. The result of these three relentlessly recurring influences in a pulsating stream, operating in a localized segment of an elastic distensible vessel, is a vicious circle: greater lateral pressure → greater widening of the stream diameter → lessened velocity of flow → greater lateral pressure → greater widening of the stream diameter → lessened velocity of flow, etc. As a result, dilatation is insidiously progressive once these hydraulic forces are brought into play.

A supremely important factor involved in the dilating effect of these minute but relentless hydraulic stresses imposed upon a vessel wall

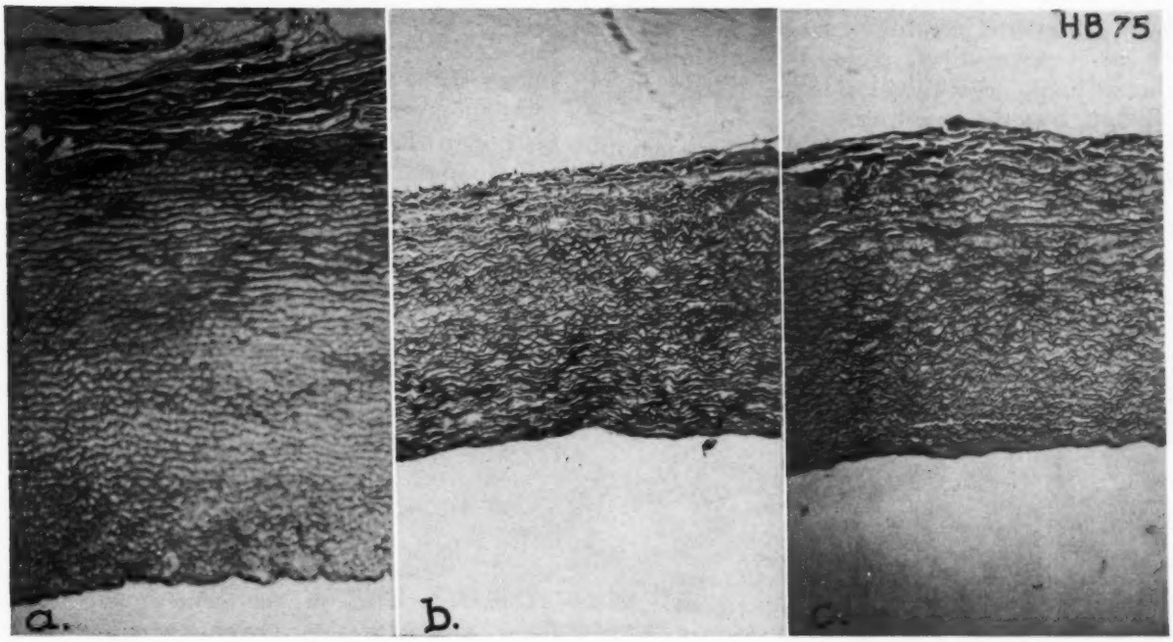

FIG. 12. Photomicrographs of aortic wall of specimen in Figure 11*a*. (*a*) Above aneurysm; (*b*) at aneurysm; (*c*) below aneurysm.

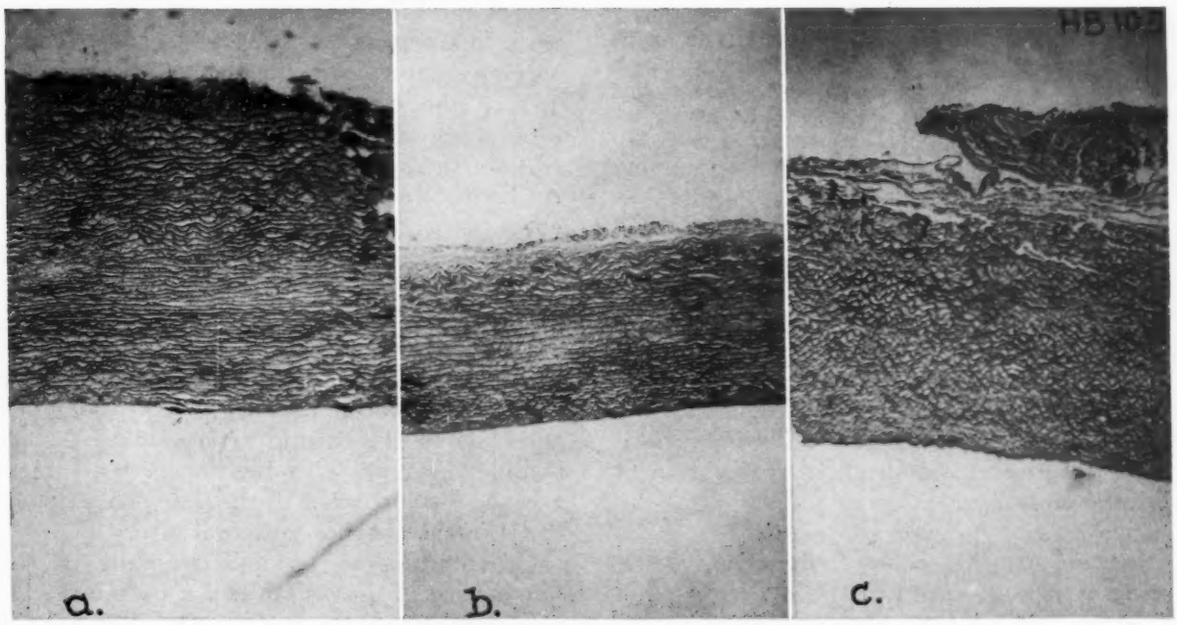

FIG. 13. Photomicrographs of aortic wall of specimen in Figure 11*b*. (*a*) Above aneurysm; (*b*) at aneurysm; (*c*) below aneurysm.

by stenosis is the extraordinary phenomenon of *structural fatigue*, long known for its peculiarly destructive and critically important qualities in the physical world of metals. Unevenly distributed in the zone of turbulent flow are waves of high pressure alternating with low pressure, resulting in impacts or shocks of high local stress against minute areas of the limiting vessel wall. These frequently repeated, inexorably recurring, impacts of high stress against the vessel wall produce structural fatigue and eventually distention of the yielding elastic wall. As the points of stress beyond the stenosis vary in position and intensity from moment to moment and are diffusely distributed in diminishing degree throughout the zone of increased

lateral pressure, so are the resulting points of strain correspondingly diffusely distributed in diminishing degree resulting in a tapering dilatation of the vessel wall.

Clinical observations: These experimental observations strongly suggest that the initiating

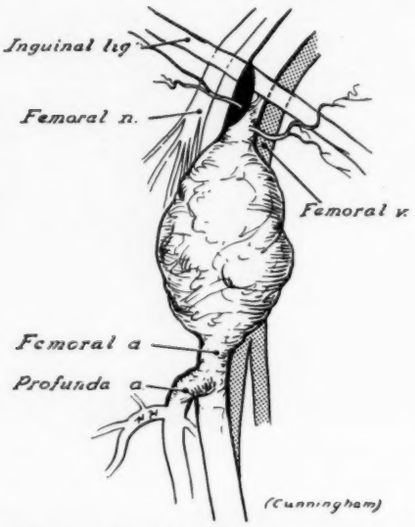

FIG. 14. At operation for a fusiform aneurysm of the common femoral artery, a definite localized obstruction caused by an arteriosclerotic plaque was present just proximal to the aneurysm.

factor in the development of an aneurysm may be stenosis of the arterial lumen imposed by segmental atherosclerosis, or by localized fibrosis, through which a narrowed stream flows with increased velocity into a still normally distensible distal segment or into a segment weakened by disease, thus setting the stage for the play of the hydraulic forces so effective in producing localized dilatation beyond a stenosis. Indeed, the following clinical example furnishes significant corroboration of this assumption.

Sixteen months before admission, while lifting a heavy weight, patient C. B. (aged fifty-two) felt a momentary burning sensation in the right femoral area. A small lump subsequently appeared in the same area and seemed to remain stationary until approximately four months before admission when it began to increase slowly in size. Coincident with this there had been pain and limping when he walked for any distance greater than three to four blocks.

Physical examination revealed a blood pressure of 164/98 and a pulse rate of 88. Below the right inguinal ligament was a pulsating mass approximately 2 cm. in diameter. The right popliteal artery was easily palpable but the pulses in the foot were absent.

At operation on March 9, 1954 (by Dr. Frank Gerbode), the aneurysm was found to involve the common femoral artery and to arise just below Poupart's ligament, extending downward for 4 to 5 cm. Its wall was thin and arteriosclerotic. Just proximal to the aneurysm, the femoral artery was narrowed by a thick arterial plaque which lay on the posterior surface. (Fig. 14.) The aneurysm extended to within 1 cm. of the bifurcation of the common femoral artery. It was excised and a child's aorta was introduced as a homograft. Right lumbar sympathectomy was performed. Examination on the day of dismissal showed excellent pulses in the foot.

Closely related in the general subject of aneurysmal dilatations are the two problems of how and why they occur at certain sites in the circulatory bed. Kampmeier's arresting studies[3] reveal the following distribution: Of 594 thoracic aneurysms, 214 were located in the ascending aorta, 203 in the transverse arch, 147 in the descending arch and 30 in the descending aorta.

A startingly similar distribution was recorded by Boyd:[4] In a larger series of 3,443 thoracic aneurysms, 1,675 occurred in the ascending aorta, 1,106 in the arch, 506 in the descending aorta and 156 in the thoracic aorta. Of equal interest is Kampmeier's study[3] of a large series of consecutive aneurysms seen at the Charity Hospital in New Orleans: 1,038 were located in the thoracic aorta, only 95 in the abdominal aorta, 12 in the carotid artery, 10 in the innominate artery, 3 in the subclavian artery, 2 in the iliac artery, 2 in the pulmonary artery and 1 in the anterior vertebral artery.

Contributing to an understanding of the significance of these data is the postulate suggested by our studies on collateral circulation[5,6] that at any given point in the vast unique network of branching elastic arteries the systolic pressure is just sufficient to overcome the resistance of the peripheral bed distal to it. Thus the systolic thrust in the ascending aorta is great enough to overcome the resistance of the entire peripheral bed, whereas the arterial pressure in the radial artery is sufficient only to overcome the peripheral resistance distal to it, and so on down to the smallest arteriole.

The height of the systolic thrust to which a vessel is subjected is accurately reflected in the thickness of its wall.

In the production of aneurysms great importance is assigned to suddenly increased intravascular tension resulting from precipitate and violent physical exertion or emotional stress. Such increased tension will produce its greatest strain at the site of maximal systolic pressure against maximal peripheral resistance, namely, in the ascending aorta. This is the site also most frequently weakened due to spirochetal invasion, and hence the site where a systolic bulge in the wall is most likely to occur. Once begun, this abnormal distention tends to increase in accelerating tempo and degree; the thinned vessel wall yields more readily to the lateral pressure of each systolic thrust and is weakened progressively as greater dilatation and correspondingly greater thinning occur. As this localized dilatation develops, the normal smaller aorta proximal to the dilatation becomes a relatively narrow vessel from which blood is ejected during systole at a higher velocity as compared with the reduced velocity in the dilated segment distal to it, thus bringing into play the hydraulic stresses associated with stenosis. The potency of these forces is greatly augmented by sudden increases in blood pressure and velocity of flow. Hence, unusual physical or emotional stress may subject the aortic wall already weakened by disease and by abnormal distention to a sudden increase in the play and potency of these hydraulic stresses, resulting in a further ballooning out of the vessel wall into a sacculated or fusiform dilatation. Also, the suddenly increased strain imposed by these stresses may result in a tear in the altered and thinned media with the development of a dissecting aneurysm.

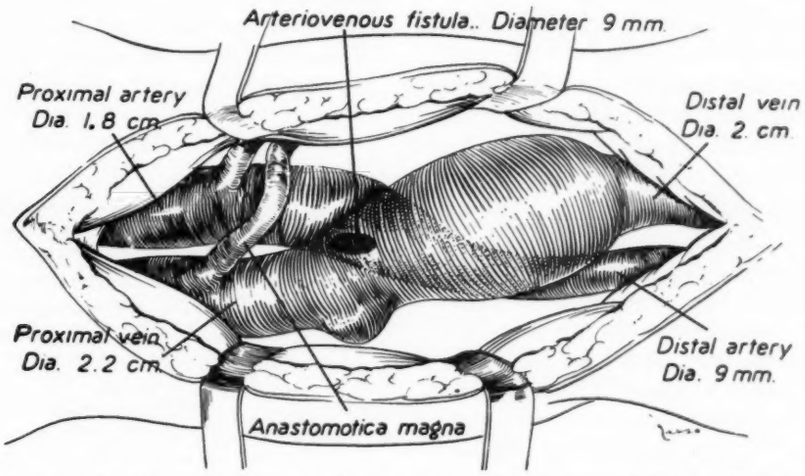

FIG. 15. Fusiform dilatation of the vein occurs almost invariably just distal to an arteriovenous communication which serves as a stenosis and which initiates the hydrodynamic forces responsible for this dilatation.

It is not just a coincidence that 70 per cent of Kampmeier's thoracic aneurysms and 80 per cent of Boyd's thoracic aneurysms were located in the ascending aorta and arch. Here the maximal systolic thrust against the maximal resistance of the distal peripheral bed combine to make the ascending aorta and arch most vulnerable to the increased tension caused by physical exertion or by emotional stress, with particularly disastrous results if part of the aortic wall in this area is weakened by syphilis or by atherosclerosis. In the descending thoracic aorta where the distal peripheral resistance has been greatly diminished, and where the systolic thrust with each heart beat is correspondingly diminished, the incidence of aneurysmal dilatation is greatly reduced as shown in both the Kampmeier and Boyd series. The low incidence of aneurysms in the abdominal aorta as compared with thoracic aneurysms (95:1038 in Kampmeier's series) is explicable on the same basis, as is also the incidence of only thirty aneurysms in the lesser arteries in this same series.

The potential power of these hydraulic stresses produced by a real or relative stenosis deserves greater recognition and respect. It may help to explain the destruction of bone and

surrounding structures that occasionally accompanies the development of a thoracic aneurysm. It explains most satisfactorily the aneurysmal dilatation of the vein usually seen at the site of an arteriovenous fistula where the arterial stream under high pressure and high velocity is ejected through a relatively narrow abnormal communication into the widened bed of the accompanying vein. (Fig. 15.) Here the peripherally directed arterial stream clashes head on throughout the cardiac cycle against the returning venous stream, producing a marked turbulence of flow, as revealed by the characteristic continuous thrill and bruit at the site of the fistula.

The occasional aneurysmal dilatation of the pulmonary artery[7] in the presence of a patent ductus may also be ascribed to the hydraulic forces liberated when the arterial stream under high pressure and high velocity is ejected from the narrow ductus into the pulmonary artery of lesser pressure, again with the production of turbulent flow throughout the cardiac cycle as disclosed by the continuous thrill and bruit of the patent ductus.

It is highly probable also that the sudden cerebral hemorrhage attributed to the rupture of a small aneurysmal dilatation or "berry" aneurysm of the cerebral vessels has its inception in a precipitate increase in the hydraulic stresses within the aneurysm just distal to the relatively narrow normal artery which accompany the heightened pressure and increased velocity of blood flow incident to emotional stress or physical exertion.

Sudden death due to rupture of a large thoracic or abdominal aneurysm may follow a sudden increase in blood pressure and pulse rate incident to emotional or exertional stress, both of which produce an increase in velocity of flow through a stenosis and an increase in the play of hydraulic forces in the aneurysmal dilatation distal to the stenosis, resulting in a marked increase in lateral pressure against the thinned out dilated wall.

These and other similar phenomena in the human vascular system may find their proper explanation in the application of the hydraulic laws that govern the pulsatile flow of fluid through an elastic vessel subjected to real or relative narrowing by congenital abnormalities, by degenerative processes or by accidental injuries.

They give stern warning also of the need to employ in the vascular surgery of children methods of end-to-end anastomosis that permit growth at the site of union as the child grows, thus avoiding future stenosis, and of the necessity of applying methods of suture in all vascular surgery that prevent or minimize the development of constriction at the site of anastomosis, either by puckering at the time of operation or by cicatricial contracture as healing progresses.

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DISCUSSION

Wm. P. Longmire, Jr. (Los Angeles, Calif.): The observations of Drs. Peniston and Holman concerning the hydrodynamic factors in the production of aneurysms have been most interesting. Certainly the authors deserve most favorable comment for their pertinent observations concerning a rather common clinical entity, which many people have observed and wondered about, but whose explanation has remained obscure until these experiments were performed by Holman and his associates.

It is also interesting that Halsted should have been so fascinated by this particular subject as far back as 1918 and that little more has been contributed to the understanding of this condition until the present observations. In referring again to the original articles of Halsted, which Holman and Peniston cited, certain of the conclusions are most interesting in light of the present observations: (1) A partially occluded artery, abdominal

aorta or innominate subclavian artery may dilate distal to the site of constriction. (2) The dilatation as circumscribed has been greatest when the lumen of the artery was reduced to one-third or perhaps one-fourth of its original size. (3) When the obturation has been slight in amount, dilation has not been observed. Of seven cases of complete obstruction there was a moderate degree of dilatation in three and none in four. (4) Complete or partial occlusion of the thoracic aorta may be followed by dilatations central to the point of constriction. (5) When the lumen of the aorta is considerably constricted, the systolic pressure may be permanently so lowered and the diastolic pressures so increased that the pulse pressure may be diminished by one-half. (6) The experimentally produced dilatations and the aneurysms of the subclavian artery in cases of cervical rib are probably not due to vasomotor paralysis, trauma or sudden variations in pressure. (7) The abnormal whirlpool-like play of blood, which is mentioned by the authors in the relatively dead pocket just below the site of constriction, and the lowered pulse pressure may be the chief factors concerned in the production of the dilatation.

These observations are particularly interesting in light of the conclusions which Dr. Holman and his associates previously reported regarding the experimental production of post-stenotic dilatation. Drs. Holman and Peniston have stated that a mass of fluid ejected through a narrow and limited constriction under high velocity strikes against a more slowly moving mass of fluid distal to the stenosis and results, first, in the conversion of high kinetic energy into high potential energy or lateral pressure and, second, in the lateral deflection of the rapid stream and even in a complete reversal in its flow. This clash of opposing streams produces eddies of alternating high and low pressures whose repeated impacts over a prolonged period against an elastic wall are capable of inducing structural fatigue and distention of that wall.

I wish to present examples of a few cases which illustrate clinically some of the principles explained by these experimental and theoretic observations.*

The first is an aortogram produced by inserting a catheter through the brachial artery down into the aorta. One can see moderately severe coarctation of the aorta with a very small patent ductus arteriosus and with moderate post-stenotic dilatation of the distal aorta.

The second example produced by a similar technic in an older patient demonstrates a somewhat larger opening in an area of coarctation with even greater dilatation of the post-stenotic aorta.

Another is an artist's conception of the anatomic arrangement of a recently explored patient showing moderate dilatation of the proximal aorta with a point of complete occlusion at the site of coarctation and a small non-distended aorta distal to this point. Undoubtedly this is largely a result of the complete occlusion which occurred at the site of coarctation in contrast to the two previous examples where there was only partial occlusion. This is also explained by the observations of the present authors.

Finally, this illustration, which shows the end results of extensive so-called multiple congenital arteriovenous fistulas involving the forearm, is taken from a case of a somewhat different type but we believe it illustrates the same principle. Here the large dilated pulsating vessels can be seen coursing throughout the forearm. In this slide are presented specimens of the subcutaneous tissue, certain large vessels which were removed at operation and also a segment of the ulna showing the cavitation produced by the dilatation of large vascular areas inside the bone. Injection with a radiopaque solution after excision of the vessel at operation shows multiple areas of constriction and dilatation, and again illustrates in a different-sized vessel certain of the principles alluded to in the present paper. In the first place the wall of this vessel is not typically that of a normal medium-sized artery but has more the character of an arterialized vein, so that as pointed out it is more susceptible to the influences of dilatation. The blood flow in the arteries to such an extremity is far greater than that in the vessels of a normal arm; therefore greater forces are available to produce aneurysmal dilatations. In addition, there are stenotic areas in various portions of the vessel with dilatations of the wall beyond these multiple points. This I believe to be an expression of the same principle which Drs. Penniston and Holman have emphasized.

May I again thank the authors for their excellent presentation of this fundamental explanation of a most fascinating clinical disorder.

* Dr. Longmire illustrated his discussion through the use of slides.

Surgical Management of Lesions of the Thoracic Duct

THE TECHNIC AND INDICATIONS FOR RETROPERITONEAL ANASTOMOSIS OF THE THORACIC DUCT TO THE HEMIAZYGOS VEIN

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FOR over three centuries clinicians have observed that the escape of milky fluid from the thoracic duct system has produced chronic disabling disease or death, yet until recent times general apathy has existed regarding all treatment except that of the cervical region. In the past decade, however, successful management of lesions in the dorsal portion of the thoracic duct has been accomplished, although those below the diaphragm are still almost totally neglected. The author's studies reveal that subdiaphragmatic thoracic duct fistulas are not uncommon and may be responsible for leakage of chyle up into the chest or down into the femoral region of the thigh. On the basis of study of eight personal cases and a review of hospital records on sixteen others, the formulation of a definite plan of treatment for each region has been made. The location of the fistula is considered one of the most important considerations. In the management of abdominal accumulations of chyle the role of the anastomosis of the thoracic duct to the hemiazygos (or azygos) vein is described. The technic and indications for the operation devised by the author are presented.

INCIDENCE

The true incidence of all types of chylous fistulas is not known. For the most part reports have been isolated, dealing mainly with cervical and thoracic lesions. Little mention is made of diseases of other portions of the duct system. As early as 1901 Allen and Briggs[1] reported fourteen cases of injury to the thoracic duct in the cervical region. DeForest[12] collected thirty-one cases of operative injuries to the cervical portion of the thoracic duct in 1907. Chylothorax was first reported in detail by Quincke[31] in 1875. By 1912 Zesas[42] recorded eighteen cases. In 1938 Shakelford and Fisher[33] recorded forty-two cases, and in 1946 Loe[25] increased the number to sixty-two. During the past eight years over twenty additional reports have been made.[2,7,9,16,19,27,33] It is interesting to note that in military surgery the first reports were apparently made in World War II[4,25] when two isolated cases were recorded. However, five cases from the Korean conflict have been reported from a review of 2,811 chest casualties.[37] One reason that so few cases have been reported as a result of warfare may be found in the fact that the thoracic duct is close to the vital structures in the neck, mediastinum and abdomen, especially the great vessels, so that direct injury to the duct usually results in a fatal wound.

The author has been able to collect eight cases from his personal experience and sixteen from the case records of chylous fistula in three hospitals.* The clinical data of these twenty-four cases are briefly outlined in Table I. In reviewing the locations of the various lesions it will be noted that the two largest groups are those of the thorax and abdomen. (Table II.) However, in the group with chyle extravasated both above and below the diaphragm, the responsible lesion is probably subdiaphragmatic. If this is true, the abdominal lesion is the most common (thirteen cases). With the present-day interest that is now being focused on lesions of the thoracic duct, it will be found that these cases are not as rare as has been heretofore supposed.

ANATOMY

The anatomy of the thoracic duct has been the subject of numerous investigations,[10,22,26,27,32] dating back to the original

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description of "arteries containing milk of Erasistratus" originally given by the Italian anatomist, Aselli, in 1622. The thoracic duct is a thin vessel varying in diameter from 2 to 6 mm. which extends from the cisterna chyli upward along the anterior surface of the bodies of the vertebrae to empty into the venous system at the angle of junction between the left subclavian and internal jugular vein. In studying the varied manifestations of the extravasation of chyle one must take into account the afferent branches of the thoracic duct which bring the chyle to the cisterna chyli. Apertures in the walls of these afferent ducts may lead to chylous extravasations which may collect not only in the abdomen but also in the thoracic or femoral regions. Chyle from the lacteals passes into tiny lymphatic channels to the mesenteric lymph nodes and then through one or more well defined intestinal trunks or afferent ducts. In addition, two branches from the lumbar lymphatics unite with the intestinal trunks to form the cisterna or receptaculum chyli on the level of the second lumbar vertebra. There is considerable variation in the number and course of the afferent branches to the cisterna chyli. There are very few valves in the short converging afferent ducts and none in the cisterna itself which is a thin-walled sac measuring about 7 by 35 mm.

Emerging from the cisterna chyli are one and sometimes two branches of the thoracic duct. Although the earlier writers believed that the occurrence of the right and left thoracic duct was common, Meade[27] reports that in 78 per cent of the cases a single duct was present. The thoracic duct leaves the abdomen through the esophageal hiatus and continues between the aorta and the azygos vein to the level of the fifth thoracic vertebra where it angles to the left to enter the superior mediastinum. It passes behind the arch of the aorta and the left subclavian artery to reach the thoracic inlet. In the neck it crosses the subclavian artery to enter the venous system at the angle of junction of the left subclavian and internal jugular veins. A right thoracic duct forms as a branch of the left thoracic duct and enters the right subclavian vein as it joins the internal jugular. There is no question, from a large number of dissections that have been made, that there is a tremendous variation in the formulation and distribution of the main and tributary channels of the thoracic duct system. In addition to the pathway mentioned, collateral afferent branches have been found to drain in the azygos and hemiazygos veins and even into the renal veins. It is important to know the common pattern and to realize that there are many variations in the formation of the ducts. Only in this way can one appreciate the possible varied pathologic pattern and plan out surgical treatment.

PHYSIOLOGIC CONSIDERATIONS

The thoracic duct has the important functions of carrying the chyle to the venous system as studied by many investigators.[5,15,18,21,38] Seventy per cent of the ingested fat enters the blood stream by this means. The fat content of chyle varies with the fat content of the diet. However, some of the fat absorbed by the lacteals goes directly to the branches of the portal veins and then to the liver. As a filtrate, chyle contains other constituents of the blood in percentages similar but smaller to that of blood serum. The protein content is less and does not parallel the diet. It varies from 1 to 6 mg. per 100 cc. The salt content is similar to that of lymph, and chyle flows at the rate of 60 to 90 ml. per hour. This is influenced by intestinal peristalsis, the pulsation of adjacent arteries, coughing, straining, respiratory motion, bending and stretching. In addition to the contraction of the muscular coats of the thoracic duct, the syphon action of the blood flowing into the left innominate vein promotes the flow of chyle into the venous system.

Chyle may be distinguished from non-chylous effusion by the fact that the specific gravity is over 1.012. The fat content varies from 1 to 3 per cent or more. Many microscopic fat globules are present. The fat is ether-soluble and can be stained with the usual fat dyes. The accumulation of fluid is rapid, up to 4,000 cc. per day. The fluid is bacteriostatic and resists putrifaction. Although lymphopenia has been described in the circulating blood due to excessive loss of lymph, we have not observed it uniformly in our cases and do not believe it is a constant diagnostic feature of the disease. A layer of cream forms on the chylous fluid on standing. On the other hand, non-chylous effusion shows no cream layer or microscopic fat globules; specific gravity is below 1.012, the fat content is below .4 per cent and the fluid tends to form very slowly.

TABLE 1

Case No.	Age (yr.) and Sex	Diagnosis	Clinical Problem	Treatment	Result	Comment
1	74,M	Chylous cervical fistula; metastatic bronchogenic carcinoma to cervical region; surgical injury to duct	Chylous cyst of neck developed following biopsy of cervical nodes; cyst drained copious amounts of chyle	Patient refused further surgery; pressure dressings unsuccessful; deep x-ray therapy unsuccessful	Died	Simple ligation of cervical portion thoracic duct would have been treatment of choice
2	35,M	Tuberculous, cervical lymph nodes; surgical injury to cervical portion of thoracic duct	Laceration of large thoracic duct in course of excision of tuberculous lymph nodes	Ligation of large thoracic duct in cervical region	Cure	Cervical ligation with good result because of adequate collateral circulation
3	65,M	Carcinoma of esophagus; surgical injury to dorsal portion of thoracic duct	Division of thoracic duct in course of excision of carcinoma of thoracic esophagus	Ligation of large thoracic duct in dorsal region	Cure	Adequate collateral circulation with ligation of dorsal portion of thoracic duct
4	65,F	Carcinoma of lung; surgical injury to dorsal portion of thoracic duct	Dorsal portion of thoracic duct torn in excising carcinoma of right lung	Ligation of dorsal portion of thoracic duct	Cure	Adequate collateral circulation in dorsal region
5	56,M	Chylothorax; chyloperitoneum; questionable etiology	Spontaneous development of chylous fluid in pleural and peritoneal cavities for 22 yr.	Aspiration only; surgery refused	Unimproved	Retroperitoneal lesion of duct likely; thoracoabdominal exploration indicated
6	41,F	Chylothorax; chyloperitoneum (retroperitoneal); neurofibroma compressing thoracic duct	Persistent chylothorax, 9 mo.	Anastomosis of hemiazygos vein, afferent branch of thoracic duct to cisterna chyli	Cured	Anastomosis successful; indicated because of location of lesion (see report of case)
7	12 days, F	Chylous ascites; etiology unknown	Chylous ascites increasing since birth, 12 days	Laparotomy; no cause for chylous ascites found	Cured	Subsided spontaneously; operation unnecessary; probable rupture of small branch of afferent ducts
8	52,M	Chyloperitoneum; retroperitoneal lymphoblastoma	Chylous ascites for 3 mo.; 12,000 cc. fluid aspirated at one time	Peritoneoscopy, no lesion seen; deep x-ray therapy without result; laparotomy with attempt drainage of chyle into abdominal wall by plastic tube	Died	All treatments failed; chances of successful ligation or duct-to-vein anastomosis are remote
			Hospital Cases			
9	31,M	Blunt injury to chest; chylothorax; cerebral concussion	Chylothorax, bilateral, after auto accident, 2 wk.	Repeated aspirations	Cured	Success with aspirations only; bilateral chylothorax
10	22,M	Blunt injury to chest; chylothorax; cerebral concussion	Chylothorax after auto accident, 1 mo.	Repeated aspirations 7 wk.; thoracotomy with attempted ligation of thoracic duct; decortication indicated but not done due to patient's poor condition	Improved	Failure of thoracentesis; probably allowed patient to get in too poor condition for operation; surgery only partially successful due to multiple fistulas; may have needed retroperitoneal exploration
11	83,M	Carcinoma of esophagus; chylothorax	Surgical injury to thoracic duct during esophagectomy	Repeated aspirations	Died	Chylothorax contributed to postoperative shock
12	27,F	Chylothorax; lymphosarcoma	Lymphosarcoma proved 6 mo.; chylothorax by inguinal node biopsy	Aspirations; deep x-ray therapy	Unimproved	Temporary improvement after deep x-ray therapy
13	23,M	Chylothorax; Hodgkin's disease	Persistent chylothorax	Aspirations; deep x-ray therapy	Unimproved	Failure of deep x-ray therapy
14	37,M	Chylothorax; bronchopneumonia; cause: ?infection, ?coughing	Persistent sterile chylothorax, diagnosed "empyema"	Drained by open drainage	Cured	Drained with erroneous diagnosis of "empyema"; healed in 6 mo.
15	52,M	Chylothorax; chyloperitoneum; heart failure; bronchopneumonia (terminal)	Persistent chylothorax; persistent chyloperitoneum for 2 yr.	Aspirations; laparotomy with insertion of plastic tubes to drain chyle to abdominal wall	Died	Cause not found; retroperitoneal exploration of thoracic duct indicated

TABLE 1 (*Continued*)

Case No.	Age, (yr.) and Sex	Diagnosis	Clinical Problem	Treatment	Result	Comment
16	37,F	Chylothorax; chyloperitoneum; chylous fistulas, femoral region	Persistent weeping of chyle from femoral region, right thigh, for 22 yr.	Exploratory laparotomy; biopsy: dilated lymph channels	Unimproved	Retroperitoneal exploration indicated; probable congenital lesion (see report of case)
17	45,M	Chylothorax; chyloperitoneum; lymphosarcoma	Persistent accumulation of chyle in thorax and abdomen	Exploratory laparotomy; biopsy, lymph nodes; deep x-ray therapy	Died	Failure of deep x-ray therapy; retroperitoneal exploration not done
18	60,M	Chylothorax; chyloperitoneum; lymphosarcoma	Persistent collections of chyle in thorax and abdomen	Aspirations; deep x-ray therapy	Died	Failure of deep x-ray therapy
19	10,F	Chylous effusion in mesentery with cyst formation	Swelling of abdomen, 7 yr.	Laparotomy; excision of mesentery containing collection of chyle, usually regarded as "chylous cyst of mesentery"	Cured	Congenital lesion; extravasation of chyle into mesentery from small afferent branch; had not ruptured into peritoneal cavity
20	66,M	Chyloperitoneum; hypertensive heart disease	Swelling of abdomen, 7 mo.	Repeated aspirations	Died	No autopsy; cause unknown; chyloperitoneum contributed to heart failure
21	32,M	Chyloperitoneum; "toxic" cirrhosis of liver	Chylous accumulation in peritoneum, 3 yr.	Aspirations; laparotomy; glass button in peritoneum and abdominal wall	Unimproved	Cause of chyloperitoneum not found; retroperitoneal exploration indicated
22	71,M	Chyloperitoneum; adenocarcinoma of stomach	Chylous accumulations of fluid in abdomen, 3 mo.	Aspiration; laparotomy, carcinoma of cisterna chyli	Died	Chyloperitoneum with advanced cancer in region of cisterna chyli
23	26,M	Chyloperitoneum; acute appendicitis	Symptoms of acute appendicitis	Laparotomy; appendectomy; chylous fluid aspirated at time of appendectomy	Cured	Incidental finding of chyle in abdomen; probable rupture of small duct which healed spontaneously; no treatment indicated
24	48,F	Chyloperitoneum; adenocarcinoma of ovaries	Swelling of abdomen, 2 mo.	Aspiration	Unimproved	Probable invasion of thoracic duct with advanced carcinomatosis

ETIOLOGY

Since the majority of articles on lesions of the thoracic duct deal with individual case reports, there is a scarcity of extensive reviews of the various pathologic conditions resulting in chylous fistulas. Most reports deal with trauma.[27,33,42] After reviewing our cases and certain reports from the literature,[3,6,39] the following comprehensive outline can be tabulated:

I. Traumatic:
  A. Blunt injury (non-penetrating)
    1. Stretching or tearing of the duct without osseous fracture
    2. Associated with fracture of the vertebra, ribs, or clavicle
    3. Extreme flexion of spine with adhesions about the duct
  B. Penetrating wounds
    1. Stab wounds
    2. Gunshot wounds
    3. Explosive fragments
  C. Surgery
    1. Cervical (excision of lymph nodes)
    2. Thoracic (esophagectomy, mediastinotomy)
    3. Abdominal (sympathectomy or radical lymph node excision)

II. Extrinsic compression:
  A. Benign tumors without invasion of the duct (neurofibroma)
  B. Malignant tumors, external pressure of enlarged lymph nodes (lymphosarcoma, carcinoma)
  C. Inflammatory nodes (tuberculous and other infections)

D. Scars resulting from inflammatory disease

III. Intrinsic lesions:
A. Obstruction of lumen from
1. Malignant tumor
2. Filariasis
B. Perforating lymphangitis
C. Thrombosis of left subclavian or innominate veins
D. Aneurysm of duct
E. Cirrhosis of liver
F. Tuberculosis
G. Congenital malformation

IV. Idiopathic:
(Cases in which the cause of the fistula has not been determined)

In Table III, depicting the etiology of cases in the author's series, trauma, malignant disease and undiagnosed lesions are listed in almost equal frequency. Surgical injury was most common in the trauma group. However, a wide variety of traumatic conditions is listed in the literature. Although penetrating injury and severe blunt injury are logical causes of fistula of the thoracic duct, it is surprising to find that even rather minor trauma such as simple hyperextension of the spine may result in fistula of the thoracic duct. It is thought that adhesions holding the duct to the vertebra may so fix the vessel that it tears on stretching over a prominent intervertebral disc or exostosis.

TABLE II
LOCATION OF THORACIC DUCT FISTULA
(AUTHOR'S SERIES)

Location	No.
Cervical	2
Thoracic	8
Thoracic and abdominal	5
Thoracic, abdominal and femoral	1
Abdominal	8
Total	24

Investigations into the non-traumatic causes of chylous fistulas have not been frequent. Washburn[39] examined the thoracic duct in 119 consecutive autopsies and found eight cases in which the duct was involved in a disease process. Four of these were carcinomatous involvement, and two were due to thrombosis, one to suppuration and one to perilymphadenitis. Others[29] have stressed the importance of clots in the subclavian or jugular veins as the cause. This mechanism may offer explanation for some of the so-called idiopathic cases in which no etiology has been found. In the author's series tumors and unknown causes were present in about equal proportion. The large number of cases listed in Table II as "unknown" is due to the fact that neither surgical exploration of the retroperitoneal or mediastinal tissues nor autopsy was performed to demonstrate the possible etiology.

TABLE III
ETIOLOGY OF THORACIC DUCT LESIONS
(AUTHOR'S SERIES)

Etiology	No.
Surgical injury	5
External trauma	2
Benign tumor	1
Malignant tumor	7
Infection	1
Unknown	8
Total	24

DIAGNOSIS

The fundamental point in the diagnosis, of course, is to make certain that the fluid being extravasated is chyle. One does not need a number of complicated chemical tests.[23] The presence of free fat droplets as seen on the stained specimen under the microscope, the extraction of stained fat with ether and the rapid extravasation of the fluid are satisfactory criteria for the presence of chyle. The signs and symptoms of chylous extravasation vary according to the regions involved (cervical, thoracic, abdominal and femoral) and are best discussed separately by these regions. Fig. 11A depicts the sites of accumulation of extravasated chyle.

Cervical. Entrance of chyle into the cervical region usually follows trauma or some operation in which injury to the cervical portion of the thoracic duct was undetected. Swelling occurs above the clavicle which is fluctuant and obviously a collection of fluid. This can usually be distinguished from an abscess by the lack of local signs of inflammation (redness, tenderness and heat) and the lack of general systemic

response to this collection of fluid. Aspiration yields a milky fluid which may at first be taken for purulent exudate. However, it contains no organisms on smear and is sterile on culture. Following the aspiration and decompression of the cyst-like structure, the swelling recurs overnight, much to the surprise and discomfort of the surgeon. This rapid accumulation of fluid may cause the patient considerable distress. If the aspirations are repeated, the soft tissue over the collection of chyle will usually break down and a fistula will result. This is exceedingly troublesome, for it is hard to believe how much fluid will drain through this route. Dressings must be changed constantly. The patient's annoyance is paralleled by the physician's frustration with ineffectual attempts to control the leakage of chyle by pressure dressings.

Thoracic. In the thoracic region the diagnosis presents a more difficult problem. The signs and symptoms are those of rapid accumulation of fluid in the pleural cavity. Three striking clinical features have been described:[23] (1) the latent period before symptoms occur, (2) the rapid extravasation of chylous fluid after aspiration and (3) the progressive dehydration and emaciation which frequently is eventually fatal. Symptoms do not develop until a large amount of fluid accumulates in the pleural cavity. In traumatic cases there is usually a lag of three to seven days[23] but it may be as long as several months or even longer. In nontraumatic cases it is difficult to estimate the exact interval between the beginning of the extravasation of chyle and severe symptomatology. In Case 6 of the author's series the patient had had symptoms for nine months prior to the first aspiration. When the accumulation of fluid is sufficient to collapse the affected lung, there is dyspnea; and finally the usual signs of shock develop from the displacement of the mediastinum and the partial obstruction to the superior and inferior venae cavae. Once this mechanical effect of displacement of the mediastinum and collapse of the lung are relieved by aspiration, the patient shows dramatic improvement. The rapid reaccumulation of fluid causes a repetition of the foregoing signs and symptoms. If the chylous fluid is mixed with blood, it may be mistaken for hemothorax. If free of red blood cells, empyema may be simulated. However, careful analysis of the fluid, as described previously, and the extreme rapidity of the accumulation will point to the diagnosis. Eventually, however, loss of weight, dehydration and inanition, reflecting loss of fat and protein, may present the picture of a wasting disease such as cancer or tuberculosis.

Abdominal. Chyle collects at first in the retroperitoneal tissues adjacent to the lesion in the abdominal branches (both afferent and efferent) of the thoracic duct. This collection may be considerable without producing symptoms other than vague abdominal discomfort. From this retroperitoneal location chyle may be accumulated under such pressure that tiny fistulas develop in the peritoneum with the pouring out of fluid into the peritoneal cavity. Presumably if the peritoneum has sufficient tensile strength to resist the rupture, the fluid may on occasion extend up into the chest or through the aortic or esophageal hiatuses; or, more rarely, along the femoral vessels through the fossa ovalis and the saphenous opening into the femoral region of the thigh. The escape of chyle into the peritoneal cavity results in the gradual formation of chylous ascites. A latent period of a variable length of time takes place here as in chylothorax. Sooner or later sufficient fluid accumulates in the abdomen to force the patient to seek medical attention. The diagnosis of cirrhosis of the liver is usually entertained until the true character of the disease is determined by careful identification of the fluid as chyle instead of ascitic fluid. Peritonitis may be suspected because of the creamy character of the fluid, and cardiac failure with ascites is also confused with chyloperitoneum. In heart failure and cirrhosis the accumulation of fluid is much slower than with chylous extravasation. In the late stages loss of weight, inanition, etc., take place. As stated previously, the true diagnosis can be made with the proper tests for chyle.

Femoral. On rare occasions, as exemplified by Case 16 in the author's series, the fluid not only may leak into the peritoneal cavity but also may appear as swelling in the superficial tissues of the femoral region. The chyle escapes from the abdomen, presumably along the femoral vessels, through the fossa ovalis, coming out under the skin through the saphenous opening. From there it diffuses to spread down the thigh. Multiple fistulas develop in the skin so there is the weeping of chyle from these various openings. It was of interest that the chyle also worked its way into the peritoneal

and pleural cavities. When the leg would cease to drain, the fluid would accumulate in the abdominal and pleural cavities with abdominal distention and dyspnea from the pleural effusion. However, most of the time the chylous drainage was so profuse from the thigh area that the patient had to wear a colostomy bag to prevent soiling of her clothes. Although we have been unable to find a similar case in the literature, it is probably true that others have been observed. However, the important point again is first the proper identification of the draining fluid as chyle, and second an inquiry into the site of the chylous fistulas. Because of the unusual findings, Case 16 will be reported in some detail:

Case 16: A thirty-seven year old housewife complained of draining sinuses in the right leg of twenty-seven years' duration. For at least twelve years there had been recurrent right-sided chylous effusion which apparently occurred when the right thigh did not drain adequately. The patient entered the hospital complaining of weakness, chills and fever which were occurring weekly. At a number of different institutions she had been treated for filariasis with arsenicals and by x-ray therapy for suspected lymphoblastoma. Neither of these diagnoses was proved.

On physical examination the patient was a pale, thin white woman, obviously chronically ill. Significant findings included deviation of the trachea to the left and signs of a massive effusion in the right pleural cavity. Examination of the heart was essentially negative. Blood pressure was 120/82. The liver edge was felt 4 cm. below the costal margin; it was tender and there was moderate shifting dullness in the abdomen. The right upper thigh was swollen with many draining blebs over which the patient was wearing a colostomy bag. Laboratory studies revealed the following: hemoglobin 12.0 gm., white blood count 14,800, 86 per cent neutrophils. The A/G ratio was 2.8:2.0. The fluid aspirated from the chest had a cream layer, fat droplets on microscopic examination and a high fat content (3 gm. per cent). Chest x-ray showed a right-sided pleural effusion. Wassermann and Kahn tests were negative. It was believed that there was a mass in the right lower quadrant of the abdomen, and exploratory laparotomy was performed on July 5, 1946. Upon opening the peritoneal cavity through a lower midline incision numerous adhesive bands were found to bind the peritoneal surfaces of the intestines together. A number of dilated lymph vessels and small saccules containing chyle were noted in the mesentery. The mass in the right lower quadrant was found to be a conglomeration of dilated lymphatic trunks and saccules. Biopsy was taken and the specimen was described as a "white cystic structure 3 by 1.25 cm. in diameter. The cyst contained milky fluid. Microscopic diagnosis was: Chylangiectasis of mesenteric lymphatics."

The patient recovered uneventfully from the operation and was discharged to the outpatient department for further aspirations.

It seemed obvious in reviewing this case that there was an abdominal retroperitoneal chylous fistula which was draining both down into the thigh and up into the right chest. Whether or not this represented some unusual congenital malformation or an actual inflammation of the afferent branches of the thoracic duct is not known because a retroperitoneal dissection was not done. The extensive amount of intraperitoneal adhesions made the exposure difficult, yet the question remains that perhaps one of the definitive procedures discussed in this paper could have been performed. Unfortunately the patient moved to a distant city shortly after this and no follow-up was possible.

TREATMENT

In the older literature the consensus was to avoid surgical interference and to allow "nature to take its course," aided by general supportive care. This included decreasing the amount of chyle by limiting the fluid and fat intake, maintenance of nutrition by high caloric and protein diets, and by the administration of chyle by vein, rectum or nasogastric feedings. That such measures are not effectual is typified by the fact that the mortality in series in which this regimen was carried out varied from 41 to 53 per cent.[23,33] In addition, deep x-ray therapy has been advised to control the leakage of chyle, particularly when a malignant disease is thought to be the cause of the chylous fistula. Klepser,[19] however, has reported failure of x-ray therapy to stop chylous extravasation in a case of lymphosarcoma. We have failed to find any improvement in four of five of our cases in which deep x-ray therapy was given for malignant disease; in the fifth case the improvement was temporary

and slight. Furthermore, inquiry reveals that the roentgen therapists in our community have had little success with deep x-ray therapy.

We believe there is definite evidence that the postponement of surgical treatment is no longer justified. Because of the fact that extravasation of chyle in each of the four regions previously presented has special problems, the treatment for each of these regions is discussed separately.

Cervical. At the beginning of this century considerable experience was gained in managing injuries to the duct occasioned by the excision of tuberculous lymph nodes in the cervical region. In 1901 Allen and Briggs[1] reported the injury to the duct in fifteen instances, with fourteen recoveries after ligation. Deansley[11] in 1903 recorded the successful implantation of a severed thoracic duct into the jugular vein. However, he believed simple ligation was satisfactory and implantation should be used only as an "alternative procedure." He quoted Kocher[20] and Treves[36] as advocating ligation of the duct. Zesas[42] in 1912 listed the treatment as (1) tamponade, (2) ligation, (3) repair and (4) implantation. Of these methods only the last three were successful. Since these early reports the cervical thoracic duct has been ligated many times. We believe the procedure is indicated in external trauma or surgical injury to the duct. We have successfully performed cervical ligation in one instance (Case 3). Conceivably, implantation of the duct into the jugular vein might be indicated in cervical obstruction of the duct where a poor collateral circulation was present. This situation is rare and purely hypothetic. In Case 2 of our series the duct was divided during the removal of carcinomatous lymph nodes. The injury to the duct was not detected at the time of operation and a huge cervical collection of chylous fluid developed with ultimate fistula formation. Because the patient refused further surgery, deep x-ray therapy was given. However, this in no manner diminished the escape of chyle. Hence ligation appears to be the treatment of choice when simple repair of the duct cannot be effected, even though malignancy is present.

Thoracic. Up until recent times the only treatment of chylothorax was aspiration of chyle to relieve the mediastinal and pulmonary compression, and general supportive measures. Surgical suture or anastomosis of the dorsal portion of the thoracic duct was considered impossible and even fatal. In 1943 Peet and Campbell[30] ligated the thoracic duct presumably divided at the time of sympathectomy. However, the patient died on the operating table. Chylous fluid was being given intravenously at the time and the autopsy report reveals no definite cause of death. This and other reports of fatalities following the intravenous administration of chyle would lead us to believe that this method of giving chyle is a dangerous procedure and has no place in the modern therapy of thoracic duct lesions now that successful surgical measures have been developed.

Loe[25] was apparently the first surgeon to effect an operative cure with chylothorax by ligation of the duct in the cervical region. He exposed the jugular and subclavian veins by dividing the clavicle. Lampson[21] in 1948 successfully ligated the dorsal thoracic duct, deliberately entering the pleural cavity from the left side. Since then a sufficient number of cases have been reported to attest the practicability, safety and the efficacy of the thoracic ligation of the dorsal portion of the thoracic duct. In each of these cases extravasation of chyle was stopped and apparently the collateral circulation of lymph was sufficient to permit the chyle to enter the venous system. Identification of the thoracic duct and its tributaries may be aided with the use of lipophilic dyes, as advised by Klepser[19] and others. Particularly efficacious was the administration of a green food coloring known as D-C No. 6, a few grams of which will amply color more than 200 cc. of fat such as olive oil or butter. If this fat is given to the patient approximately four hours before surgery, identification of the duct will be greatly facilitated.

Meade et al.[27] advocate aspiration of chyle and closed drainage before resorting to surgery. Phrenic paralysis[28] has been successful in stopping the flow of chyle in chylothorax in a number of occasions. These minor procedures are not always effectual but may be tried prior to thoracotomy or when thoracotomy is contraindicated. We believe their greatest chance for success is to be found soon after the chylothorax is established. There is no question that ligation has proved successful. We have had two personal cases in which this was done. Both were injuries to the thoracic duct inadvertently made during thoracotomy (Cases 3

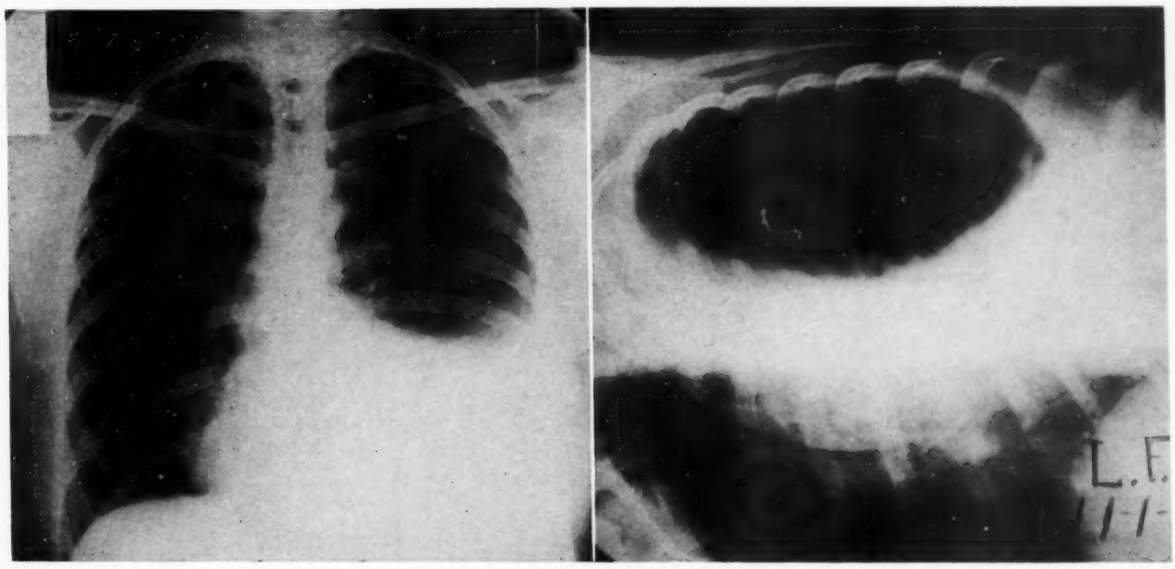
and 4). In both instances the laceration of the duct was identified and promptly closed. The collateral circulation was adequate in each case. Of course, if repair of the duct is possible, it is indicated.

Hodge and Bridges,[17] using experimental animals, worked out the technic of implantation of the severed dorsal thoracic duct into the azygos vein. They have successfully applied this technic on a human patient. There is no question that physiologically it is sound to perform anastomosis rather than ligation. We believe, however, that for the perforation of the duct in the thoracic region in most instances simple ligation is all that is necessary because of the rich collateral circulation. The ligated duct should be observed very carefully. Certainly it should be tried first. If there is no rupture of the ligated duct due to back pressure and lack of collateral circulation, this simple technic will suffice.

Abdominal. There is no question that successful methods of treatment for lesions of the thoracic duct in the cervical and thoracic regions have been employed on a sufficient number of cases so that these methods can be considered firmly established. However, no such success has been achieved for lesions of the thoracic duct below the diaphragm. The problem is here complicated by a more difficult approach to the thoracic duct system, and also by the fact that attempts at treatment have been made without regard to what has been learned about therapy for lesions above the diaphragm. If the chylous extravasation occurs in the mesentery, it forms a cyst-like structure which may be simply excised, providing the diagnosis is made before there is rupture into the general abdominal cavity. Example of such a case is found in the author's series, Case 9. In most instances little if any surgical attack has been made to correct the underlying cause of extravasation of chyle. Tubes have been placed in the peritoneal cavity to allow the fluid to drain out of the peritoneum into the abdominal wall where it may be absorbed. This method has produced no decrease in the amount of chyle or the patient's symptomatology. It was employed in three cases in the author's series without any beneficial effect whatsoever. In the abdominal extravasation of chyle the fistula may be in the afferent ducts, the cisterna chyli or the efferent ducts. If the afferent ducts are involved, the operation of choice under certain circumstances is anastomosis of the thoracic duct to the hemiazygos vein, or azygos vein, as is described in the following case report:

Case 6: A forty-one year old white woman sought medical care because of increasing dyspnea of nine months' duration. The onset was insidious with gradual progression and increasing weakness. Cough had been present for three months, with pain in the left chest and loss of 10 pounds in weight. Chyle had been aspirated from the left chest on a number of occasions during the previous month. The past history was non-contributory. On physical examination the patient was a fairly well nourished adult woman who was seen to be chronically ill. The significant findings were those of a huge collection of fluid in the left chest, and shift of the heart and mediastinum to the right. The abdomen showed slight diffuse tenderness but no mass or signs of acute inflammation. Thoracentesis yielded 1,000 cc. of creamy fluid with all the characteristics of chyle. A postero-anterior upright x-ray following the thoracentesis (Fig. 1A) showed considerable amounts of fluid in the left lower space. The lateral decubitus film (Fig. 1B) revealed no tumor of the pleura or left hilum. Bronchoscopy and esophagoscopy for suspected tumor showed no lesion. Laboratory studies revealed the following: hemoglobin 16.5 gm., red blood count 5,140,000, white blood count 8,800, polymorphonuclears 65 per cent, lymphocytes 29 per cent and eosinophils 6 per cent. An upper gastrointestinal x-ray series demonstrated no abnormality.

On November 24, 1951, a left transthoracic thoracoabdominal exploration was performed; 60 cc. of olive oil were injected through a Levin tube into the stomach before the operation. Incision was made in the seventh intercostal space on the left, and after entering the pleural cavity 2,000 cc. of chyle were aspirated. Of interest was the fact that there was no pleural reaction and that in spite of a nine-month history the lung could be readily expanded by the anesthetist. Chyle was seen to be oozing out from three places above the diaphragm, posteriorly, and two places near the root of the lung. (Fig. 2.) The mediastinal pleura was opened and it was obvious that the chyle was oozing from below the diaphragm. A horseshoe incision was made in the diaphragm, a procedure which we usually employ for exposure of



1A 1B

FIG. 1. Case 6. Preoperative films. A, postero-anterior view after aspiration of 2,000 cc. of chyle and air injection. B, lateral decubitus projection shows no tumor of pleura or left hilum.

the upper abdomen.[8] There was only a slight amount of chyle in the abdominal cavity; however, it was steadily pouring forth from the retroperitoneal space. By dividing the left crus of the diaphragm an excellent view of the retroperitoneal space behind the aorta was gained. (Fig. 3.) Two nodules about 2 by 1 by ¾ cm. in diameter could be seen; they were firm in consistency, grossly appearing to be tumor tissue. Oozing of chyle came from the caudad extremity of the more inferiorly placed nodule. After excision of these nodules biopsy showed we were dealing with a benign neurofibroma. Two small ducts extravasating chyle, which were thought to be afferent branches to the cisterna chyli, were ligated. The cisterna chyli could not be identified and was thought to have been destroyed by the tumor. In ligating a larger third branch this vessel began to distend with chyle so much that it was paper thin; and if we had allowed the ligature to remain in place, it would undoubtedly have ruptured. The ligature was hastily loosened and the situation reviewed. It became apparent that the ligature was compressing this main duct in the same manner as the tumor. Furthermore the rapid dilation of the duct to the point of rupture revealed that the collateral circulation of the adjacent lymph channels was not adequate. It was thus certain that decompressing anastomosis to a vein was necessary if the patient was to be cured. Fortunately the hemiazygos vein was close by and it was possible to mobilize the duct so the two structures could be joined. A side-to-side anastomosis was made with a simple running stitch of No. 5–0 arterial silk. Bleeding was controlled from a vein by loop ligatures from either side of the anastomosis which were loosened when the anastomosis was done. Upon completion of the anastomosis the duct filled with chyle and the vein with blood. (Fig. 4.) It was possible to milk blood up into the duct and to see it move back into the vein again, proving the patency of the anastomosis. Since there was no further leakage of chyle, the diaphragm and chest wall were closed in layers with interrupted cotton sutures. The phrenic nerve was crushed at the diaphragm. Pleural drainage was effected by means of a Foley catheter placed in the eighth intercostal space and connected with a closed drainage system. Figure 5 depicts location of chyle in the thorax and abdomen.

The patient's postoperative course was uneventful. There was no further evidence of leakage of chyle. When last seen three years and two months after surgery she was well and working. Figure 6, an x-ray taken at that time, shows the left lung completely expanded.

Pathologic report on the resected tumor was as follows: "Tumor is characteristic of a neurofibroma and is made up of irregular whorls of interlacing strands and bundles of elliptically shaped cells. These have small

ovoid or elliptical nuclei which show slight variability in morphology. There are no mitotic figures. Scattered through the tumor are numerous thin walled, endothelial lined vascular spaces. In places occasional follicle-like aggregates of lymphocytes are seen. (Fig. 7.)

"On one section a large thick walled, endothelial lined, vascular-like structure is seen to have the wall composed of longitudinal and

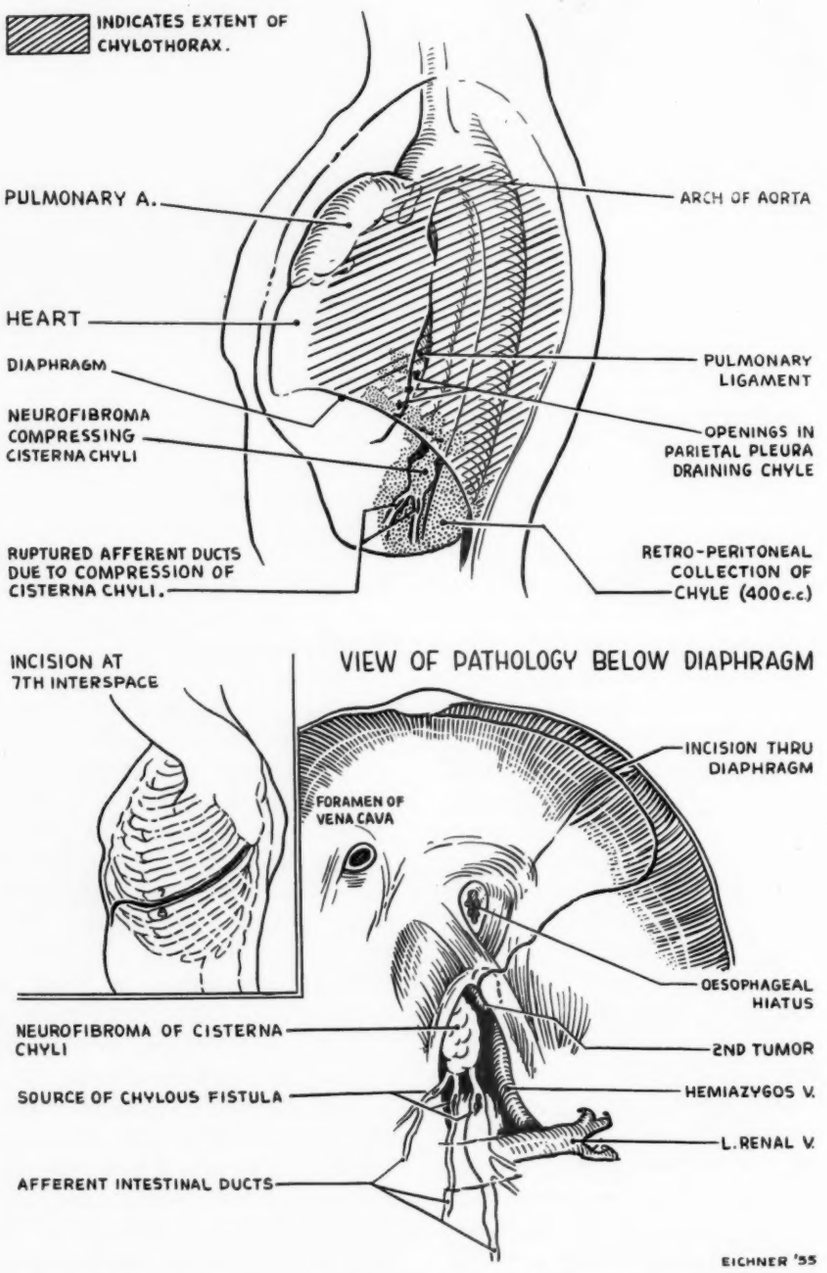


FIG. 2. Case 6. Illustration of findings in left pleural cavity. After aspiration of 2,000 cc. of fluid, chyle seen oozing from mediastinal and diaphragmatic pleura from below the diaphragm.

FIG. 3. Same case. Neurofibroma rests on cisterna chyli obstructing the afferent lymphatic ducts which have ruptured and are leaking chyle.

circular muscle bundles. (Fig. 8.) The muscular layers are conspicuously thickened and are interspersed with a somewhat edematous fibrocollagenous stroma. The lumen is compressed and relatively empty. This structure resembles a compressed branch of the cisterna chyli which was not identified on the gross specimen."

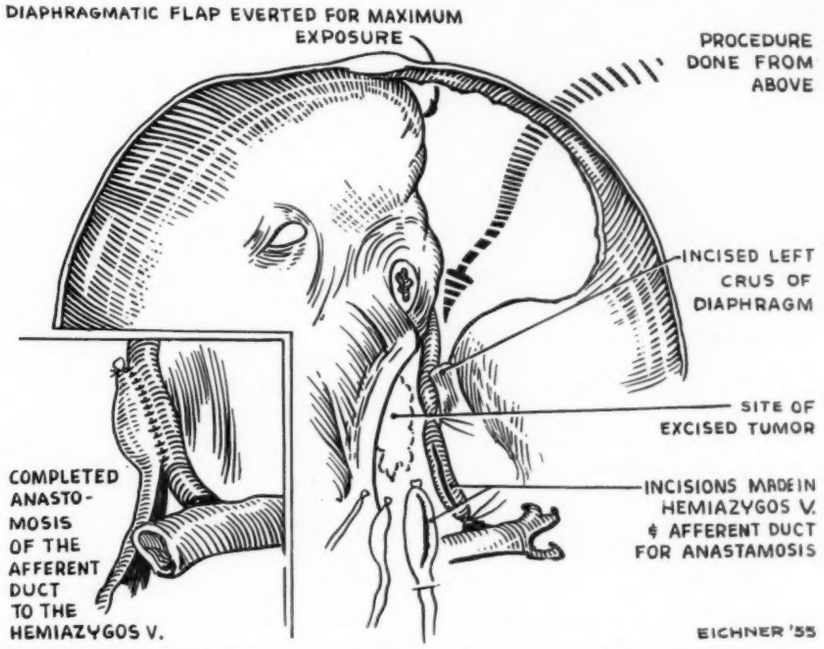

DIAPHRAGMATIC FLAP EVERTED FOR MAXIMUM EXPOSURE
PROCEDURE DONE FROM ABOVE
INCISED LEFT CRUS OF DIAPHRAGM
SITE OF EXCISED TUMOR
INCISIONS MADE IN HEMIAZYGOS V. & AFFERENT DUCT FOR ANASTAMOSIS
COMPLETED ANASTO-MOSIS OF THE AFFERENT DUCT TO THE HEMIAZYGOS V.
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FIG. 4. Case 6. After ligation of afferent ducts, largest branch dilated with chyle under pressure and was on point of rupture. Decompressing anastomosis to hemiazygos vein made.

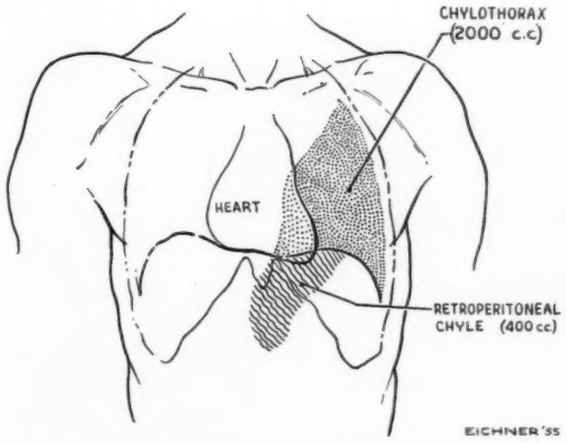


FIG. 5. Case 6. Location of collection of chyle in thorax and retroperitoneal space from rupture of afferent ducts to cisterna chyli.

Femoral. The dissection of chylous fluid from the abdomen down to the femoral regions of the thigh, as mentioned previously, brings up a number of interesting problems. We have been unable to find any case in the literature where a chylous femoral fistula was present in which direct attack on the thoracic duct system was made. It is unfortunate that one patient in our series (Case 16), after having had an exploratory laparotomy, moved to a distant part of the country and has not been traced. We believe that the management of the fistulas in the thigh must depend upon the location and control of the chylous extravasation in the branches of the abdominal portion of the thoracic duct system. It could involve either the afferent ducts, the cisterna chyli or the efferent ducts. Thus the method of treatment outlined in the previous paragraphs would be applicable here. If there were sufficient collateral circulation, simple ligation might be satisfactory; however, it is reasonable to assume that whenever possible anastomosis of one of the afferent ducts to the hemiazygos or azygos veins would be the procedure of choice.

COMMENTS

Results of the various forms of treatment employed in the author's series are outlined in Table IV. One is impressed that in fifteen of sixteen patients treated with repeated aspira-

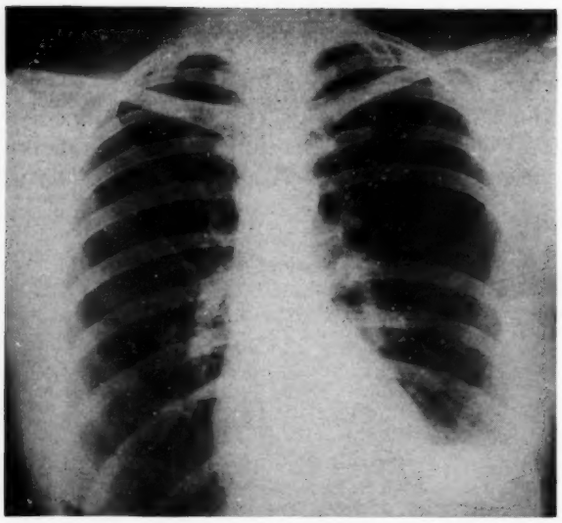
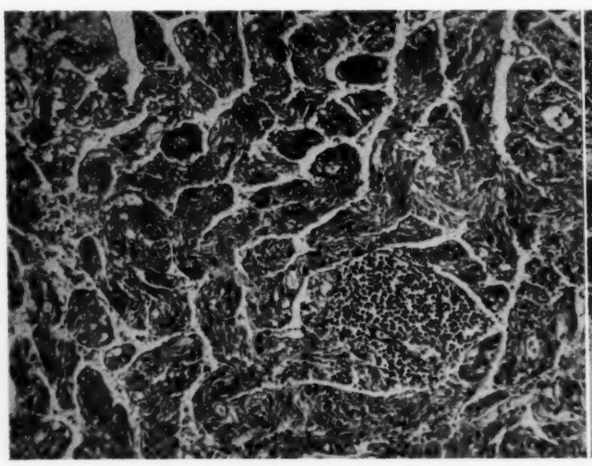


FIG. 6. Case 6. X-ray of chest three years and two months after operation. Left lung expanded; no further accumulation of chyle.

tions there is no cessation in the leakage of chyle. It was successful in only one case. Drainage of the chyle from the abdomen into the subcutaneous tissues was of no avail in three cases and x-ray therapy failed in five of six instances to decrease the chylous extravasation. This is contrasted with the success gained in the ligation of the thoracic duct and the anastomosis of the duct to the hemiazygos vein in which complete success was attained. With cervical and thoracic lesions it seems clear that direct ligation of the thoracic duct above and below the fistula is the procedure of choice. This should be done promptly in the cervical region; however, in the thoracic region it is certainly permissible to have a trial of aspiration and/or closed drainage, or phrenemphraxis before resorting to surgery. Surgeons should not delay, however, until the patient gets into poor physical state with depleted proteins, etc., before resorting to surgery. In Case 10 the patient's condition was too poor for the operation to be satisfactorily completed. For the treatment of chylous fistula in the abdominal region, ligation may be carried out for lesions above the cisterna chyli. However, when the collateral circulation of the thoracic duct is inadequate, anastomosis to a vein is indicated.

TABLE IV
SUMMARY OF TREATMENT

Therapy	Unimproved	Improved	Cured	Died
Aspiration	16	..	..	3
Drainage to subcutaneous tissues	3	..	..	1
X-ray therapy	5	1	..	4
Ligation	..	1	3	..
Anastomosis, duct-to-vein	..	..	1	..
Spontaneous cure	..	..	2	..
Miscellaneous	..	..	2	..
Totals	24 (Failures)	2	8	8



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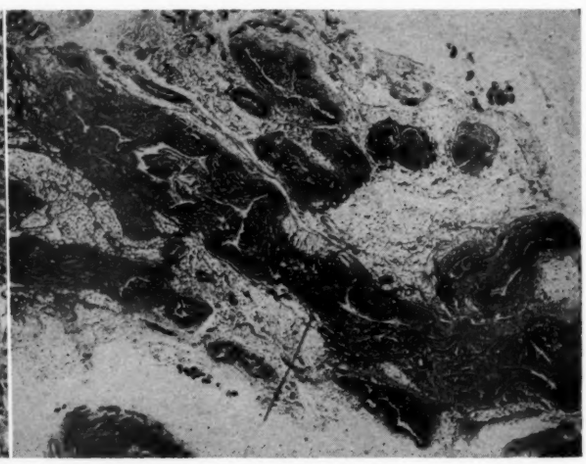

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FIG. 7. Case 6. Microscopic section of tumor. Elliptically shaped cells arranged in whorls typical of neurofibroma. Follicle-like collection of lymphocytes is present.

FIG. 8. Same case. Microscopic section showing vascular structure composed of longitudinal and circular muscle bundles having the appearance of a compressed branch of the cisterna chyli.

Several methods have been reported for uniting the thoracic duct to a vein. Deansley,[11] as early as 1901, and later Harrison[16] (Fig. 9) implanted the duct through the proximal end of the divided external jugular vein in the cervical region. More recently, Hodge and

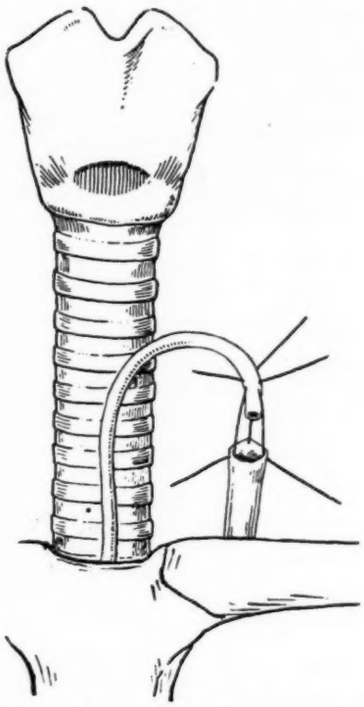


FIG. 9. Technic of implantation of thoracic duct into external jugular vein, employed by Harrison.[16]

Bridges,[17] after considerable experimental work, reported a method of implanting the divided thoracic duct into the undivided azygos vein in the mid-dorsal region. (Fig. 10.) Recently Demarchi et al.[13] reported the use of tiny gold intraluminary tubes to anastomose the thoracic duct to an intercostal vein. The lumen of this vein is so small it is far more preferable to use the azygos or hemiazygos vein. We believe it is not important what method is used to join the thoracic duct to a suitable vein, providing the anastomosis remains patent. The important point is the principle of employing anastomosis when the collateral circulation of the lymph channels carrying the chyle is insufficient. This may be due to (1) congenital abnormalities, (2) ligation of collateral tributary ducts or (3) destruction of collateral tributary ducts by disease. Under these circumstances anastomosis and not ligation is the procedure of choice, providing mural repair cannot be done. In the femoral region collections of chyle have their

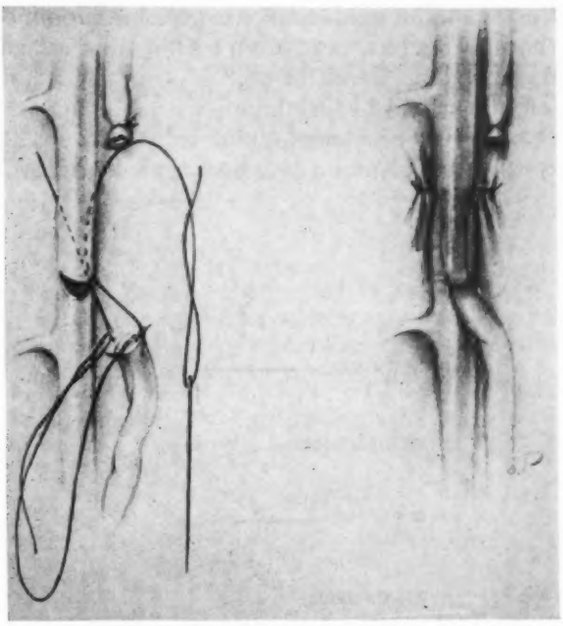


FIG. 10. Technic of implantation of thoracic duct into azygos vein, used by Hodge and Bridges.[17]

origin in the abdomen; exploratory laparotomy or thoracolaparotomy is indicated for the careful investigation of the cisterna chyli and both the afferent and efferent branches. Figure 11 outlines the sites of collection of chylous fluid and the surgical treatment recommended for each site.

The mortality in our small series parallels that reported in the literature. (Table IV.) We found eight known deaths with three other probable ones since these patients had advanced malignancy at the time of discharge. Nine patients were cured so that only four cases are questionable. This gives a mortality of a probable eleven of twenty-four patients (46 per cent). In the literature mortality varied from 43 to 50 per cent.[19] One of the reasons for the high mortality in our series may be that in thirteen of the cases there was no attempt to perform any surgical measure such as those outlined in this communication. To be sure, in seven cases malignant tumor was diagnosed (29 per cent), so that in these cases we could not hope for permanent cure. However, cure may have been possible in the others.

SUMMARY

1. A study of eight personal cases and sixteen hospital records of thoracic duct fistula both above and below the diaphragm has been presented. In this series chyle most often formed

in the abdominal and combined abdominothoracic regions; next often was in the thoracic region alone, and rarely in the neck and femoral region of the thigh.

2. Trauma and malignant tumor proved to be the most common causes of chylous extravasation although the number of undiagnosed conditions was large.

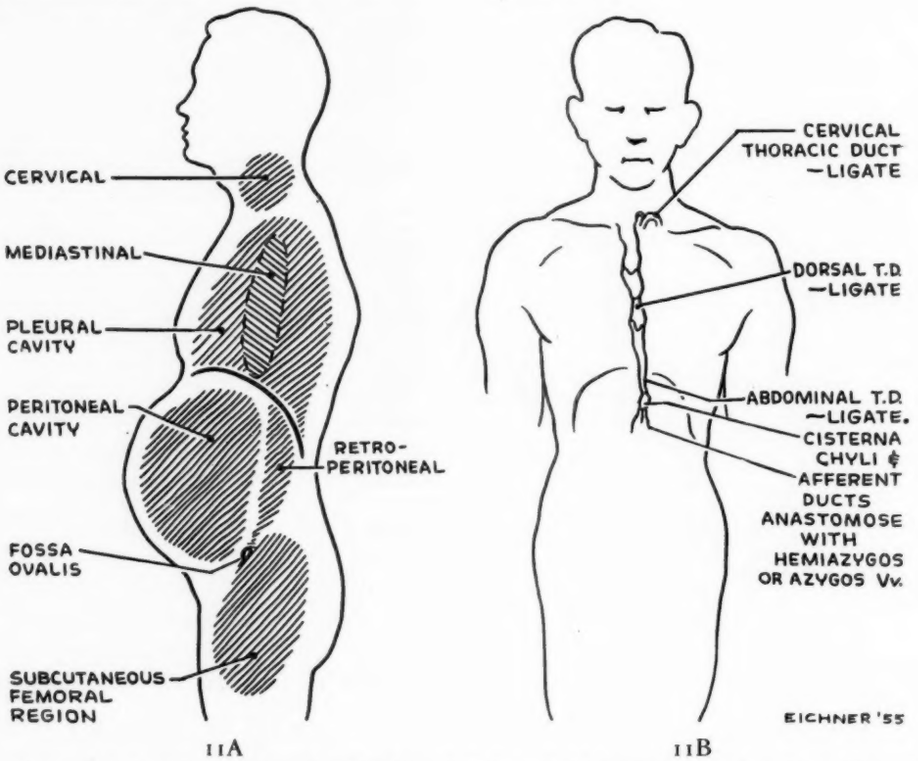
3. The location of the fistula of the thoracic duct is as important as the etiology in determining the treatment.

4. In the cervical and thoracic regions simple ligation of the duct is generally successful.

5. Below the diaphragm the treatment of chylous fistulas has been generally neglected. The indications for anastomosis of the afferent thoracic duct (and efferent, at times) to the hemiazygos or azygos veins are presented. The author's original technic of this retroperitoneal anastomosis of the thoracic duct to the hemiazygos vein is described.

6. Aspiration of chyle and/or deep x-ray therapy resulted in a high mortality, while ligation and anastomosis procedures can have a low mortality (none in our series) and a high rate of cure.

7. It is believed that the mortality and the rate of permanent cure can be greatly improved. The extension of the surgical treatment, as outlined in this communication, to lesions below the diaphragm should effect this improvement.



11A 11B

FIG. 11. A, location of sites of collection of chylous fluid in author's series. B, outline of type of operation employed by author for lesions of various levels of the thoracic duct system.

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DISCUSSION

RALPH H. LOE (Seattle, Wash.): Dr. Brewer has presented a fascinating problem in an original light that would appear to be an important step forward in the management of the obstructed thoracic duct in the abdomen. This contrasts with the accepted therapy of ligation for injuries to the duct in the chest. Dr. Brewer's experience with traumatic thoracic duct injury has certainly been extensive. It is apparent that there are now many surgical injuries to the thoracic duct resulting from the greatly increased amount of thoracic surgery being performed.

In 1946 I reported on a traumatic chylothorax which was perhaps the first cured by ligation of the thoracic duct. The chylothorax was due to a healed bullet wound which had cut the thoracic duct in the neck and had torn a hole in the pleura, permitting a continuous leakage of chyle. The rapid downhill course of the patient was successfully stopped by ligation of the thoracic duct. This duct had been ligated many times in the neck during the course of radical neck dissections without incident. I have done so once to my knowledge.

The animal work of Lee as long ago as 1922 and later by Blalock in 1937 indicated that the thoracic duct could be ligated in the chest, either high or low, without undesirable effects. Yet as far as I know this procedure was not done until 1946 for the actual treatment of a case of chylothorax. It is

interesting to know that Blalock had extreme difficulty in producing chylothorax or chyloascites in seventy-five animals in which all of the ducts possible were ligated, including resection of the receptaculum chyli. Anastomosis between veins and lymphatics in the abdomen is apparently quite extensive. It would appear, however, that there are cases in which the communications are not sufficiently great in the abdomen to prevent chyloascites. In that event the operation indicated by Dr. Brewer has a very definite place. Since 1946 there have been, as mentioned by Dr. Brewer, many cases of ligation of the thoracic duct in the chest without ill effects. Dr. Brewer's delicate anastomosis in the abdomen is a credit to his skill. Anastomosis between a vein and the thoracic duct in the neck has been done previously. However, one would not know whether the anastomosis actually functioned or not, because obliteration of the anastomosis would act in the same manner as ligation, which is well tolerated in the cervical region.

Last week Dr. Henry Harkins and I saw a patient with bilateral chylothorax and chyloascites. This had been of rather slow onset, beginning six months previously. There was also a congenital lymphangiomatous change in the left hand. There is no history of trauma except an indefinite one of having fallen down in an ice locker approximately at the time of the development of this condition. One case has been reported by Brown in which a relatively minor fall severed the thoracic duct in the diaphragmatic area with bilateral chylothorax and chyloascites. Operative intervention failed to find the location of the injury, but postmortem examination proved the severance of the duct. It would seem that Dr. Harkins' case might possibly be suitable for Dr. Brewer's intriguing thoracic duct vein anastomosis if no injury to the duct is demonstrated. I can see no other procedure which would be of any help unless there is actual severance of the thoracic duct, in which event simple ligation might be sufficient. The use of dyes and a fat meal for aid in location of the thoracic duct is a distinct operative aid.

It would seem that under certain circumstances the collateral circulation of lymph in the abdomen is not sufficient to prevent chyloascites, thus presenting a problem which has apparently been solved by Dr. Brewer's ingenious venous thoracic duct anastomosis.

Frederick L. Reichert (San Francisco, Calif.): The lymphatic system is just as extensive as the venous or the arterial. We should all keep that in mind as it is a very important system. Dr. Brewer did not tell us if it was a sympathetic type of neurofibroma. Was it or was it not? (Yes.) This is very important because we do not know if the sympathetic fibers are on lymphatics. If a dog can be given an anaphylactic shock, the arteries to the veins are contracted but the lymphatics are huge. I am quite amazed that Dr. Brewer could make such a beautiful anastomosis with the thin thoracic duct; it must have been a little thickened because it was distended partially from the constriction of the neurofibroma. Certainly the thoracic duct should be ligated if there is a leak. We see this duct sometimes when performing sympathectomy in the thorax. Several times we have had to ligate it, without any trouble. As Dr. Brewer said, giving some substance to visualize the thoracic duct very often helps to identify it. However, if you know your anatomy, you know where to pick it up.

Joseph A. Weinberg (Long Beach, Calif.): Dr. Brewer has brought to our attention an important subject on which there are vital questions still unanswered. For example, just how is lymph conveyed to the venous circulation after accidental or intentional ligation of the thoracic duct, and why it is necessary to perform an anastomosis to a vein at this low level when mere ligation may suffice at a higher level?

Injuries to the terminal portion of the thoracic duct may become more frequent in the future as the result of more frequent operations of dissection of the lymph nodes along the lower part of the internal jugular vein in connection with bronchogenic carcinoma. We have had one experience in which a fistula was created during such a dissection. The fistula closed within a few days following compression with a gauze roll over the area. The favorable outcome in this case, which is not a unique experience, is probably explained by the fact that not all of several radicles at the terminal portion of the duct are injured and enough radicles remain to maintain the flow of lymph into the venous system. Tissues should be clamped and ligated during the excision of nodes in this area in order to avoid the complication of lymphatic fistula.

Robertson Ward (San Francisco, Calif.): I am sure that many of us have had experience with interference in the thoracic duct in the neck. I am equally sure you cannot anastomose the thoracic duct, as it occurs in the neck with any vein. My experience is limited to only one case; but when I saw the thoracic duct in that neck dissection, it looked just like a blister with a thickness of one layer of pavement endothelium. I inquired among my colleagues and found that a number of them had had the experience of severing the thoracic duct in the neck. Two methods of treatment were recommended. One was to pack the area with iodoform gauze. I tried this procedure and I rise to warn against it. The patient was a youngster upon whom I performed a neck dissection for carcinoma of the thyroid, recurrent after heavy external radiation. Necrosis of the subcutaneous tissues

developed, resulting in a wide open neck which took a long time to heal. I do not know but what the necrosis developed partly as a result of post-radiation fibrosis. It is sufficient to warn against the use of iodoform packing. I would like to ask Dr. Brewer if he thinks an anastomosis such as he has described would be possible in the neck.

LYMAN A. BREWER, III (closing): Dr. Loe is a real pioneer in this field and I appreciate very much his fine discussion. Dr. Reichert has raised an important point regarding the possibilities of the type of tumor. We had considerable discussion regarding what type of neurofibroma this was. I thought perhaps that it might be a tumor of the lymphatic system, but our pathologists believed it exhibited more characteristics of nerve tissue origin. Additional opinions on these sections would certainly be welcomed.

As far as Dr. Weinberg's comment is concerned, I think it is very timely because of the present popularity of the scalenus muscle biopsy, which is an important weapon in our diagnosis of thoracic diseases. Certainly his comment of not leaving any possible vessels open in the cervical lymphatic channels is well taken, for one need not fear ligation of the duct at this level. Regarding the site of the thoracic duct fistula in my case, I should like to repeat that it was located at the level of the second lumbar vertebra. This was a retroperitoneal anastomosis and was not in the thorax. The fluid had formed quite a large pocket retroperitoneally. With the circular incision of the diaphragm we had excellent exposure of the left abdomen.

Dr. Ward's comments were of interest. We were unable in one case to control drainage successfully by pressure dressings although in some instances this may be successful. I want to make it clear to Dr. Ward that we do not recommend anastomosis in the neck. We think that simple ligation is all that is necessary due to excellent collateral circulation of lymph. I think in those patients with obstruction of the afferent branches of the cisterna chyli it would be easier to find an afferent duct that would be large enough to anastomose. If the duct were very small, perhaps the use of tiny gold intraluminary tubes would be successful in these cases. I am very pleased with the response to this paper, as evidenced by the number of discussers and their interesting remarks.

Surgical Treatment of Ulcerative Colitis

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FROM the standpoint of treatment, ulcerative colitis is one of the most perplexing conditions of the colon. The characteristics which render definitive therapy so difficult have been commented upon in virtually all of the reported series. Among these are the obscure etiology of the disease and the wide variation in clinical manifestations and symptomatology. Perhaps most confusing so far as treatment is concerned is the unpredictable response of ulcerative colitis to therapeutic measures.

In general it is a chronic disease, the natural course of which may vary greatly. It is characterized by exacerbations and remissions; in many patients the stationary level is compatible with considerable activity on the part of the host. In some cases the disease is self limiting; in others it progresses gradually and continuously. A remission may be of long duration, but a recurrence of symptoms may appear at any time. The causative factors producing a remission are largely unknown; usually a remission occurs spontaneously rather than as a result of any particular therapy. In a small percentage of cases ulcerative colitis manifests as an acute, fulminating, septic disease. Infrequently, an acute episode occurs as the initial appearance of ulcerative colitis. More often acute cases are those in which the disease has been present for some time and eventually reaches a critical stage during an exacerbation.

Primarily, chronic ulcerative colitis is a medical disease and should be treated as such. Only when complications or progressive intractability occur does the question of surgical intervention arise. It is with the relatively small group of patients for whom surgical treatment may be the therapy of choice that this report is primarily concerned, although some of the general characteristics of this disease as manifested in our series will be briefly reviewed.

From January 1937 to January 1954, 255 cases of ulcerative colitis were seen at the University of California Hospital. No increase in incidence was apparent during this period, that is, the number of cases treated each year remained essentially the same. The average age of onset was thirty-two years; seventy-two patients were under twenty-one years of age, and thirty were under eighteen. Although it is evident that the disease has a predilection for the younger age group, we have seen it at almost every age.

Despite extensive investigation the etiologic factors precipitating this condition have never been conclusively defined. Each case in our series was thoroughly reviewed for any factor, either in the environment or personality, that might have exerted an important psychologic influence on the course of the disease. In about 40 per cent of the cases some such condition was present, but there was no factor common to enough patients to be of significance in the group as a whole. It is more likely that a neurotic personality is produced by, not the cause of, the disease. Similarly, in analyzing the associated diagnoses, no significant correlation was apparent.

By the very nature of the disease almost all of the patients in our series had diarrhea, ranging in duration from a few weeks to many years and in severity to as many as twenty to thirty bowel movements a day. In the majority of cases, pus, mucus and blood were present in the stools. The principal complaint of seven patients with rather severe involvement, however, was constipation, and in other cases constipation had preceded the onset of diarrhea. The next most prominent symptom (seventy-three cases) was abdominal pain, either colicky or cramping. In some instances it was generalized in character, in others it was localized to the lower abdomen. Weight loss, amounting in some instances to as much as 40 to 50 pounds, was recorded in the majority of cases. Fever, and nausea and vomiting were the next most prevalent symptoms, while weakness, severe

malnutrition and anemia were present in a number of patients. In several cases systemic changes such as pyoderma, skin ulceration, arthritis, and ankle edema had become acute. Rectal complications included incontinence, perirectal abscess, rectovaginal and fecal fistulas, and stricture. The average duration of symptoms in our series exceeded three years, and in one case bloody diarrhea had first occurred thirty years previously.

The pathologic process involved in ulcerative colitis originates in the mucosa of the colon and is characterized by hyperemia and the appearance of small, superficial ulcerations and petechiae. During the active phases of the disease the ulceration may spread through increasing segments of the bowel, until in some instances the entire colon eventually becomes involved. In addition to this progression along the length of the colon, varying degrees of penetration through the bowel wall may occur, first into the submucosa and then into the muscularis or serosal layers. As the ulcerative areas enlarge and spread, granulation of bowel tissue takes place, resulting in the pus and blood so characteristic of the disease. With increase in size, the ulcerated areas tend to coalesce; in some cases the mucosa is completely obliterated. In other instances areas of the mucosa may remain and eventually progress to the pseudopolypoid formations often found in ulcerative colitis. Focal ulceration and necrosis may result in complete penetration of the bowel wall, leading to the perianal or rectovaginal fistulas and to abscess formation which often occur in advanced cases. Perforation into the peritoneal cavity may likewise occur in the acute phase of the disease.

During the remissive phase of ulcerative colitis, fibrous tissue forms to replace that eroded by ulceration. When the patient has experienced repeated cycles of exacerbations and remissions, this fibrotic tissue may eventually irreparably alter the haustra so that normal absorption and secretion can no longer occur. It is to damaged colon of this type that the term "lead pipe" has been applied frequently.

It is generally assumed that ulcerative colitis begins in the rectum and extends proximally, although Garlock and Lyons[5] point out that this order of progress may be more apparent than real since the rectum is more accessible to examination. In 199 of our cases report of barium enema or sigmoidoscopy or both were available. The extent of involvement in these cases was as follows: thirty-six cases (18 per cent), entire colon and terminal ileum; seventy-one cases (36 per cent), entire colon but not terminal ileum; twenty cases (10 per cent), colon distal to hepatic flexure; ten cases (5 per cent), colon distal to mid-transverse colon; twenty-six cases (13 per cent), colon distal to splenic flexure; twelve cases (6 per cent), rectum and sigmoid; twenty-four cases (12 per cent), rectum only. It should be mentioned, however, that frequently the ascending colon will appear normal in a barium study of the colon, only to be found involved along with the descending and transverse colons at the time of operation.

TREATMENT

Corticoid Therapy. Although this paper is essentially concerned with surgical therapy, it should be noted that in recent years cortisone and ACTH have been used in the treatment of ulcerative colitis with hopes of a definite action. The follow-up of many of the patients so treated, however, shows prompt recurrence of symptoms, usually with a severe exacerbation, after cessation of therapy. This result is plausible because there is no known action of these drugs on the colonic process; they may, however, accentuate it by blocking immune response, interfering with healing and masking symptoms. Patients receiving corticoids for long periods often do not do well after ileostomy with partial colectomy, and there is a tendency for shock to ensue.

Our experiences therefore suggest the following two conclusions concerning the use of corticoids in the supportive therapy of ulcerative colitis: (1) In poor risk, chronically diseased patients or those with the acute fulminating type of ulcerative colitis, the administration of cortisone is indicated, beginning with a few drops preoperatively and tapering the dosage during the few days postoperatively. (2) The use of cortisone is indicated in a patient with ulcerative colitis to tide him over some complication of the disease or any condition which may make surgery a poor choice at the time.

Surgical Therapy. Not long ago operative treatment was rarely resorted to in ulcerative colitis except as a lifesaving procedure during an acute phase or in cases which had progressed

to complete invalidism. Within the past several years, however, a trend toward increasing use of surgical therapy has been discernible in the literature. Along with the reduction in mortality that has resulted from supportive antibiotic therapy, there has been a greater awareness of the danger of delaying operative treatment in advanced cases. Another factor tending to reduce reluctance to resort to operation has been the great improvement in the technic of ileostomy and in ileostomy appliances, as a consequence of which increasing numbers of patients have been able to achieve a relatively comfortable and satisfactory adjustment.

The wide variation in clinical manifestations and the unpredictability of the course of ulcerative colitis in the individual patient make it impossible to establish rigid criteria for surgical intervention. In general, however, the conditions which were believed to warrant surgical treatment in our series correspond with those enumerated by other authors. These may be categorized as follows: (1) Permanent and irreversible change in the bowel; (2) acute local complications, such as abscess, fistula, stricture, obstruction and perforation; (3) acute systemic complications, such as arthritis and ulceration; (4) obvious failure of medical therapy to control the disease; (5) chronic incapacitation or inability of the patient to support the burden imposed by prolonged medical and hospital care; and (6) the presence of polyps or evidence of malignant degeneration.

The duration of medical treatment in our series ranged from one month to many years; the average duration was twenty-nine months. However, when repeated exacerbations of increasing severity or acute complications occur, despite all efforts at medical therapy, surgical treatment should not be delayed. Similarly, when the ulcerative process has so altered the bowel wall that restoration of normal function is no longer possible, operative therapy alone can afford some measure of relief to the patient.

Another positive indication for surgical intervention is the presence of polyps or evidence of malignant degeneration. In his review in 1952, Machella[7] found the incidence of colonic carcinoma to be 3 per cent (ranging from 0 to 7 per cent) in 6,890 cases of ulcerative colitis reported in the literature since 1944. There is almost unanimous agreement that the incidence of malignant degeneration rises sharply with the severity and prolonged presence of ulcerative colitis. Cattell[3] has noted an incidence of malignant change between 25 and 30 per cent in patients who have had the disease for eight years or more. Certainly, when carcinoma does occur in association with ulcerative colitis, there is virtually no hope of cure. For this reason and because of the evidence pointing to polyps as precursors of carcinomatous change, we believe that the presence of polyps is a definite indication for surgical intervention. In our series there were five cases of carcinoma, an incidence of 2 per cent.

It is particularly those patients who fall into the fifth category who are now being considered as candidates for surgery more often than formerly. Prolonged ulcerative colitis not infrequently imposes an intolerable economic burden on the patient. Thus, in our series several patients not only had been unable to work for extended periods but also had had to bear the expense of being hospitalized for several months, in some instances on more than one occasion. In cases of this type earlier surgical intervention may relieve the patient of an unbearable financial situation and restore long-lost earning capacity. In addition, the prognosis will be substantially better as a rule if operation can be carried out before such extreme debilitation occurs.

OPERATIVE PROCEDURES

Of the 255 cases in our series, fifty-five (21.5 per cent) were treated surgically. In general, the surgical percentages cited in the literature have ranged from 15 to 25 per cent,[1,4,5] although in some recent series as high as 40 to 50 per cent[6,10] of the patients have been operated upon. The procedures carried out in our series and the results of operative treatment are shown in Table 1.

The two patients who underwent only appendicostomy were seen during the early years of the series; appendicostomy is no longer considered adequate operative therapy in this disease. Of these two patients one was symptom-free for a four-year period, after which he was lost to follow-up. No further operation was performed during that time because of the presence of rheumatic heart disease. The other patient was last seen in 1942, two months after appendicostomy. At that time it was believed that because of a relative remission

of symptoms and a complicating psychiatric problem, further surgery was not advisable.

Ileostomy. A certain percentage of patients with ulcerative colitis will respond satisfactorily to ileostomy alone. Brown and Crile,[2] for example, reported that seventeen of ninety patients in their group operated upon prior to fall, 1949, obtained excellent results from this procedure.

In our series fifteen patients were treated by ileostomy only. (Table 1.) Of these, ten were in poor condition at the time of operation, and nine died in the postoperative period. Four patients with a follow-up extending over several years have been symptom-free after ileostomy without further operation. The remaining two patients still have some symptoms of their disease, but we believed that a more extensive procedure was contraindicated. In our opinion the factors which mitigated against further operation were the patient's advanced age, the relative mildness of symptoms and toxicity, and the limited involvement of the colon as determined radiologically.

In another instance ileostomy was performed in 1938; it was not until 1953 that evidence of reactivated colitis prompted colectomy. On the other hand, this case also illustrates the contention of most investigators that ileostomy does not cure ulcerative colitis and that continuing symptoms and further exacerbations may be experienced at any time, often within a very short period.[1,10] For this reason and the fact that polyps or carcinoma may occur in the diseased bowel, ileostomy is now generally regarded as a preliminary rather than a definitive procedure.

The ileostomy should be placed through a separate wound made in the right rectus muscle in such a position that, when finally established, it will be sufficiently distant from the umbilicus to allow easy application of an ileostomy bag. An effort is made to suture the last several inches of the terminal ileum to the anterior peritoneum to allow for fixation of this segment. We have recently utilized to good advantage the suggestion of Turnbull,[9] wherein the serosa and muscular coats of the ileum are excised and the resultant mucosal tube is sutured to the margins of the skin at the site of the ileostomy.

Colectomy. In the earlier years of our series ileostomy was usually performed as the initial operative procedure. In recent years there has been a trend toward combining ileostomy with partial removal of the colon as far down as the sigmoid as the first step of the surgical program. When positive indications for operation are present and the patient is in reasonably good condition, the procedure of ileostomy with partial colectomy allows for the removal of a large part of the diseased colon, permitting a better postoperative period in most cases. The removal of the larger portion of the colon as the initial procedure likewise permits rather easy access to the abdomen at a later stage because of absence of adhesions and extensive scarring. Since the patient's condition will improve so greatly following this procedure, because of the partial removal of the source of the sepsis, the operative treatment can then be completed at the second stage by abdominoperineal resection of the remaining colon. There will be a few patients whose poor condition will preclude a procedure of any greater magnitude than ileostomy as the initial stage, although it should be borne in mind that it is in these severely ill individuals that the partial removal of the symptom-producing colon would be of greatest benefit.

Table 1
RESULTS OF SURGICAL TREATMENT OF CHRONIC ULCERATIVE COLITIS

Procedure	Number	Deaths	Symptom-free	Improved with Symptoms	No Improvement
Appendicostomy	2	..	..	1	1
Ileostomy	15	9	4	2	..
Colectomy	27	4	20	3	..
Partial colectomy and anastomosis*	7	4	1	2	..
Limited colon resection	2	..	2	..	..
Sigmoid colostomy	1	..	..	1	..
Bowel resection	1	..	..	..	1
Total	55	17	27	9	2

* In one patient the ileum was reanastomosed to the ascending colon after ileostomy.

Total colectomy, usually performed in stages, was performed in twenty-seven patients in our series. (Table 1.) In only three cases was it possible to perform a one-stage total removal of the colon and rectum. Of these three patients,

one died on the thirty-seventh postoperative day due to pulmonary edema and bacterial endocarditis. One of the remaining two patients is completely well, while the other is still disabled because of a fistula between the ileum and the anterior abdominal wall.

It is to be admitted, as others have reported, that the one-stage total removal of the pathologic process, regardless of its location in the body, is advantageous if such is possible without prohibitive morbidity and mortality. In the presence of ulcerative colitis the removal of the disease process would prevent the loss of tremendous amounts of blood and proteins, and would minimize the absorption of toxic materials.[8] It is our feeling, however, that the one-stage procedure necessarily carries a substantially greater hazard than does the removal of the colon in stages. Moreover, the patient's condition is so improved by partial colectomy that the excision of the remaining bowel can usually be accomplished easily and with negligible morbidity and mortality. If a patient is young, is not in a fulminant stage of the disease and is in a good nutritional state, one might extend the operative procedure to include the rectum. In our own experience, however, the number of patients in this category is extremely limited.

The procedure most commonly used in our series (twenty-four cases) was the removal of the terminal ileum and the colon down to the mid-sigmoid, which was brought out as a mucous fistula. In five cases in this group the colon was removed down to a level below the pelvic floor with the proximal end of the rectum closed at the initial stage. The remaining segments of the colon and the rectum were removed either by abdominoperineal resection or merely by perineal resection if only the rectal segment had been retained at the initial stage.

Of the twenty-four patients with staged colectomies, there was one operative death and two patients have subsequently died of other causes. The remainder are either symptom-free or have minor complaints referable to the extensive scarring in the perineum or to the functioning of the ileostomy. The good results in this particular group of patients substantiate our belief that in the great majority of instances the removal of the disease process in stages is accompanied by low mortality rate and an over-all excellent recovery.

In only two cases in the series were limited colon resections possible. (Table 1.) In one of these resection of the transverse colon only was performed with end-to-end anastomosis. This patient was seen regularly in the follow-up clinic over the three years following operation, during which time she gained weight, had one to three loose stools per day, and in general did very well. In the other patient the disease was limited to the left half of the colon, allowing transverse colostomy to be performed rather than ileostomy. Although only ten months have elapsed since operation, the patient has been entirely asymptomatic.

Excision of the Rectum. In most series reported in the literature the rectum has been involved in the great majority of cases.[4,10] For this reason many authors believe that except in rare instances operative treatment of ulcerative colitis is incomplete unless the rectum is removed, and that re-establishment of bowel continuity cannot be satisfactorily achieved in a sufficient number of cases to warrant leaving a rectal stump which may cause a recurrence of symptoms.[1,4,8,10] On the other hand, Garlock and Lyons[5] believe that continued observation after colectomy is often necessary in order to determine the advisability of extirpation of the rectum. They have carried out reanastomoses in seven patients; four have remained well, one for a prolonged period.

In our series the degree of involvement of the rectum varied considerably. Only in those instances when the condition of the bowel precluded all possibility of subsequent improvement in the rectal segment was abdominoperineal resection carried out concurrently with colectomy, or as soon thereafter as the patient's condition warranted this further procedure. In general, it has been our policy in cases of less acute rectal involvement to have the patient return for systematic observation before proceeding with rectal excision.

In six cases in our series the colon was removed down to the rectum, and anastomosis between the terminal ileum and the rectum was performed. In this group there were four operative or hospital deaths; three from generalized peritonitis due to a leak at the anastomosis and one from postoperative anuria. In one of the two remaining patients the rectal segment was pliable at operation and only moderate mucosal changes were present. This patient has remained well since operation, a period of about

ten years. In the other case ileoproctostomy was performed, mainly at the patient's insistence, in the presence of severe pathologic change in the rectum. The patient has remained fairly well during a four-year period, but has required frequent cauterization of rectal polypoid excrescences, all of which have proved benign on histologic study.

In one further case ileostomy was established because of progressive symptoms. Although colectomy had been planned as a subsequent procedure, the patient recovered so well following ileostomy that upon the urging of some of the medical staff, the ileum was simply reanastomosed to the ascending colon. This procedure was carried out four years ago. The patient is presently being carried on an antiallergic regimen; and although serial barium studies of the colon and ileum show progressive changes, she is still comparatively asymptomatic.

In most cases in our series, however, removal of the rectal segment was necessary. Continued discharge of bloody mucoid material, sphincter incontinence, extensive scarring and stenosis of the rectal segment, intractable rectal fistulas, and evidence of an inflammatory polyp prompted subsequent removal of the rectum in our series.

There were two additional cases in our surgical series which cannot be classified under any of the foregoing operative categories. In one case (Table I), sigmoid loop colostomy was performed because of obstruction due to a large inflammatory mass. When the colostomy was opened, the mucosa of the proximal loop appeared to be quite normal, suggesting that the ulcerative colitis might be limited to the rectosigmoid segment. The patient has been moderately well since colostomy was performed some eight months ago. The other patient (Table I) was admitted to our surgical wards for care after having had resection of both large and small bowels elsewhere; because of his extremely poor physical and mental condition, however, further operation was deemed inadvisable, and he was released for custodial care elsewhere.

Complications of Surgery. Postoperative complications following the surgical treatment of ulcerative colitis are frequent. In our series the complication most often encountered was small bowel obstruction, which occurred in seventeen patients (35 per cent of the operative cases). (Table II.) In all but two cases the obstruction took place at the stoma site. In four cases more than one obstruction occurred at the stoma.

The balance between stenosis at the stoma and prolapse of the ileum at the stoma is indeed a delicate one. In almost every instance we have attempted to provide an opening in the abdominal wall just large enough to fit snugly around the ileum and the index finger. In spite of this, however, the incidence of stenosis was far greater than that of prolapse (six cases), suggesting that the tendency is toward establishing a smaller opening than is intended due to the relaxation of the abdominal wall by anesthesia. An additional factor, of course, is the edema which occurs not only in the layers of the abdominal wall but also in the ileum itself at the site of the stoma, thereby setting the stage for obstruction at this site.

TABLE II

POSTOPERATIVE COMPLICATIONS OF CHRONIC ULCERATIVE COLITIS

Complications	No.
Small bowel obstruction	17
Wound abscess	6
Ileostomy prolapse	6
Fecal fistula	3
Separation of suture line in bowel anastomosis	3
Intussusception	2
Perforation of bowel	1
Endocarditis	1
Subdiaphragmatic abscess	1
Mesenteric thrombosis	1
Iliac thrombosis	1

The next most frequent complication was wound abscess which occurred in six cases. In addition, there were three cases each of fecal fistulas and separation of the suture line in the bowel anastomosis. No other complication was observed in enough cases to be significant.

Results of Operative Treatment. In order to evaluate the results of operative treatment in our series, we divided the patients into the following three categories with respect to their condition at the time of the first definitive operative procedure: (1) Good: those whose physical condition indicated that the proposed operative procedure carried no greater risk than any other abdominal procedure of similar magnitude. (2) Fair: those whose physical condition suggested that the necessary procedure carried a risk three to five times that of any other abdominal procedure of similar magnitude. (3) Poor: those whose physical

condition indicated that the operative procedure carried the maximal although not prohibitive risk.

On this basis twenty-two patients in our series were considered in good preoperative condition, twenty-two were fair, and eleven were in poor condition. Of the eleven patients in poor preoperative condition, there were ten deaths in the postoperative period, or a mortality of 90 per cent in this category. Of those in fair condition, four died in the postoperative period. Particularly significant is the fact that among those patients judged in good condition at the time of operation, there were no postoperative deaths. (Table III.)

TABLE III
RESULTS OF OPERATIVE TREATMENT OF CHRONIC ULCERATIVE COLITIS

Condition	Number	Operative Mortality	Deaths Due to Other Causes	Alive
Good.....	22	..	3	19
Fair.....	22	4	..	18
Poor.....	11	10	..	1
Total.....	55	14	3	38

SUMMARY

Chronic ulcerative colitis remains a mysterious disease, the cause of which is as yet unknown. Most patients are adequately managed on a medical program. When the involvement of the colon by the pathologic process is minimal or moderate, the problem is in no way a surgical one. Certainly the disease varies greatly in duration and severity. However, when the disease has so progressed that the bowel wall is ulcerated, fibrosed and stenotic, when there is considerable bleeding, and when polypoid lesions are present in the colon, ulcerative colitis should be considered a surgical problem.

The number of patients in our series who received surgical therapy (21.5 per cent) is similar to that reported by others in the literature. Although the number of cases per year which have come to surgery has not increased, it is significant and gratifying to see that the number in good preoperative condition has been significantly greater in the past few years. By this means the mortality associated with the surgical treatment of this disease has markedly diminished.

Although an occasional patient may warrant one-stage removal of the colon for chronic ulcerative colitis, it is still our opinion that the staged removal is by far the better procedure. In recent years we have combined ileostomy with partial removal of the colon, usually as far down as the sigmoid, as the initial procedure. Ileostomy as the first stage of the operative procedure was mainly performed in the early years of the series and the mortality incident to it was excessive. Complications have been frequent, as is to be expected in the surgical treatment of such a debilitating and extensive disease process.

CONCLUSIONS

Despite the strong desire to preserve the rectal segment in any patient for whom one is responsible, it is generally agreed that if surgery is undertaken in ulcerative colitis, it should be carried to completion by the total removal of the colon including abdominoperineal resection of the sigmoid and rectum. The statement that "an ileostomy life is worse than death" is no longer tenable since modern appliances, an intelligently regulated diet, and an understanding by the patient of the type of disease involved will usually allow him to perform his customary daily routine without distress or embarrassment.

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DISCUSSION

Clayton G. Lyon (San Francisco, Calif.): I should like to thank Dr. Grimes for the privilege of reading and discussing this most interesting paper, for I think he has presented a most instructive review of the subject as illustrated by this group of patients. I observed a number of this same group over the years and I think Drs. Grimes and Boudoures have neglected to mention that in contrast to the majority of patients seen in private practice, any group collected at similar institutions will show a high percentage of complicated cases. This is further borne out in the large number of surgically treated patients who were considered to be in only fair and poor condition preoperatively.

It is interesting to note that in this group of patients the average duration of the disease was twenty-nine months. It is increasingly evident that the over-all mortality of ulcerative colitis is much higher in the first two years of the disease. The course of this disease is quite variable. More than half of the cases which are mild at the time of the first examination will improve. Patients who are extremely ill with the disease when first seen tend to become worse in a fairly large percentage of cases.

The incidence of associated carcinoma in 2 per cent of this group is rather low. It is probable that many of the earlier cases treated medically had carcinoma eventually which was not diagnosed during their active course.

With more frequent total colectomies it is likely that we will find a much greater incidence of carcinoma. Carcinoma, in addition to being difficult to recognize, is very malignant and rapidly progressive.

It is likely that once ileostomy has been elected, stage colectomy should be accepted by the patient and the surgeon should be most energetic in carrying through this plan. This has been adequately demonstrated by Dr. Grimes' presentation.

Often the patient with a well functioning ileostomy is fairly comfortable and able to return to duty before colectomy is completed. The inflamed bowel remains to act as a low grade focus of activation for arthritis or kidney and pancreatic disease.

The complications of ileostomy are most annoying. I believe that one should remove an adequate segment of terminal ileum to assure removal of the involved bowel, and perhaps a little additional mesentery to avoid leaving scarred lymphatics adjacent to the stoma. This may help to lessen the incidence of fistula and stricture.

Robert A. Scarborough (San Francisco, Calif.): I agree thoroughly and heartily with Dr. Grimes' conclusions, except for one. I believe he is approaching what we consider the optimum surgical procedure in the great majority of these patients, and that is the single, complete, one-stage procedure of abdominal colectomy, abdominoperineal resection and simultaneous ileostomy. As far as the risks of such a procedure are concerned, I have a paper now in press which reviews the operative mortality of the procedure Dr. Grimes recommends. The operative mortality of simultaneous ileostomy and subtotal colectomy now runs between 3 and 4 per cent. We have had twenty-two consecutive cases of ulcerative colitis in the past two years in which we have performed complete proctocolectomy and ileostomy as a single-stage procedure with no operative mortality. A little over half of these patients were in a state of relative remission of their chronic disease, but in the others the disease was in an acute, fulminating stage in which intensive medical therapy in the hospital failed to produce any evidence of remission. In one case the operation was performed in the presence of generalized peritonitis. We believe that even under these circumstances the entire procedure can be done, and that morbidity and mortality are minimized by this procedure since we remove all of the diseased colon and prevent any further toxemia from the disease itself.

The other matter I would like to comment on is ileostomy dysfunction. Dr. Grimes has listed intestinal obstruction as the most common complication. He had seventeen such cases, the majority of obstructions being at the level of the ileostomy. As many of you know, Drs. Turnbull and Crile in Cleveland described the mechanism of such dysfunctions as due to a serosal peritonitis on the protruding ileum which produces a physiologic obstruction. We have known for years that if we pass a catheter through the abdominal wall via the ileostomy we can relieve that obstruction. In our experience, until recently, over 50 per cent of our patients have shown some evidence of such physiologic partial obstruction, and in many of these patients we had great difficulty in controlling the excessive loss of fluids and electrolytes. We believed that we had to follow them very carefully and replace such losses. But by Turnbull's description of the technic of primary anastomosis of ileum mucosa to skin with a sleeve of mucous membrane turned back over the ileum, our last nine consecutive cases have had no dysfunction of any kind.

Leon Goldman (San Francisco, Calif.): In many of the patients reported by Dr. Grimes, cortical steroids were used for variable periods of

time before any operative treatment was instituted. I would like to say a word about the usage of these substances and the role they play in the therapy of this disease.

We have had enough experience by now to draw the following conclusions: First, patients with ulcerative colitis who are on cortisone or ACTH for long periods usually do not do well if operative treatment is instituted during that time. Second, these substances do not have any more beneficial effect on the course of this disease process in the bowel than ileostomy does. The disease process continues in the sidetracked colon. As a matter of fact, a number of articles have now appeared in the literature reporting ill effects, perforation, delayed healing, progression of infection and severe exacerbation after the discontinuance of adrenocortical substances for the treatment of this disease.

There are two instances when cortical steroids can be used to great advantage. First, in preparing the poor risk patient with chronic disease or one with acute fulminating ulcerative colitis for operation, it is helpful to administer cortisone for a very short period of time before operation and then taper it off postoperatively. Second, when a patient is being treated conservatively, cortisone may be of definite value in tiding him over a complication.

We have been disappointed in the effect of chemotherapeutic or antibiotic agents in the control of ulcerative colitis. There are some bacterial infections in which we can influence the organism and still come out ahead by giving cortisone or ACTH. In ulcerative colitis, however, we cannot cover the cortisone by the antibiotic because the antibiotics available so far do not seem to be effective. Many patients have been kept on these drugs for long periods, only to have the disease return, often in a more vicious form, when these substances were discontinued.

Charles E. MacMahon (Seattle, Wash.): Although my personal experience in this field is limited, I would like to bring up something of a technical nature that was not mentioned in performing a one-stage total colectomy. I have performed the posterior phase of the resection as the primary part of the procedure. The patient is placed in the Buie position initially and the posterior dissection completed; then in the supine position, laparotomy is performed. Having completed the posterior dissection early in the procedure while the patient is still in good physiologic balance, the excision of the colon is essentially an atraumatic® and non-shocking procedure. This has worked well for me in three total colectomies.

The second problem has to do with the preoperative evaluation of the patients with chronic ulcerative colitis upon whom total colectomy is contemplated. Why are these patients ill-prepared to approach surgery or apt to go into the hypotensive state during the course of the procedure? I think we should pay some attention to the work of Dr. Champ Lyons of Tulane and Dr. Zollinger at Ohio State on the proper preoperative appraisal. In addition to the usual preoperative blood studies, we have utilized quantitative blood volume determinations using risa. If we used the technic of quantitatively replacing the blood preoperatively, I think some of the patients who might otherwise unexpectedly develop hypotension, either during or shortly after surgery, might enjoy an uncomplicated surgical experience.

Orville F. Grimes (closing): I want to express my appreciation for the excellent remarks of the discussants. Dr. Scarborough's mortality rates are remarkable and are to be expected in his hands. Certainly it is intriguing to believe that one might perform total colectomy in one stage. I am sure that many of us, however, have had to terminate an attempted one-stage procedure just below the level of the pelvic floor. This circumstance makes the procedure a difficult one because the thickness of the rectum and its inflammatory reaction makes closure of the rectal stump hazardous. So for that reason, we have more or less terminated the resection at the level of the sigmoid colon.

We, too, have used the Turnbull procedure to good advantage recently and believe rather strongly that it has great merit in the prevention of peritonitis which so often occurs in the area around the stoma and the mesentery of the terminal ileum.

I was very happy that Dr. Goldman mentioned something about steroids. The fact that in the past decade we are receiving patients in better condition for surgery probably is due to two things: first, the greater awareness of the dangers of delay in operating on patients with extensive ulcerative colitis; and second, the preoperative preparation with steroid compounds. I agree completely with Dr. Goldman that the long-continued use of these preparations in an effort to cure the patient of ulcerative colitis is not successful.

Non-penetrating Trauma to the Heart

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I MUST first apologize for my lack of originality. In fact I have very little, if anything, to offer that is new or startling concerning the problems of non-penetrating trauma to the heart. This paper is purely the product of ignorance—my own and that of many of my colleagues.

Claude Beck,[1] presenting this subject at the American Medical Convention in San Francisco in 1934, stated that practically no one with whom he discussed the subject had heard of it. Twenty years later I receive almost the same reply. What is even more revealing, I have searched the records of the University of California Hospital, the Stanford University Hospitals and the San Francisco City and County Hospital, academically controlled by both Universities, and have been unable to find a single case of non-penetrating trauma to the heart. That is why I have taken the liberty of presenting a review of this problem.

The majority of cases of severe general or chest trauma are handled by general surgeons or members of one or more of the surgical specialties. The majority of recent articles on non-penetrating trauma of the heart have appeared in highly specialized medical or pathologic journals. In these journals the authors are mainly concerned with learned discussions on variations in P waves,[2] QRS complexes,[3] RS-T changes,[4] etc., all of which is foreign to most surgeons. This paradoxic situation may explain the general lack of knowledge concerning this subject among those who handle trauma.

With due respect to history, the first recorded case of non-penetrating trauma to the heart[5] is credited to Aluff Borch in 1676. His patient was an eight year old boy who had fallen and bruised his chest. The major complaint was pain in the chest and abdomen, and in the ensuing months the child became pale. Death occurred four months after the injury; anatomic studies revealed marked damage to the right auricle with clot formation. This is particularly interesting as, according to Osborn,[6] the right auricle is the most common site of cardiac trauma. I believe it is worth mentioning that one of the more recently reported cases of contusion of the heart was also in a child.[7] This girl, six years of age, was struck severely on the chest by a fist. She immediately turned pale and cardiac difficulties developed. Subsequent physical and laboratory findings confirmed the suspicion of contusion to the heart and treatment was instituted similar to that for coronary occlusion.

Warburg has reported on a search of the literature, collecting 261 cases of non-penetrating trauma to the heart in which survival was six days or longer, and including eighteen cases of his own.[5,8] Gore[9] is dubious of many of Warburg's cases but does accept about 40 per cent of them as presenting reasonably good evidence of the presence of traumatic heart disease. Many of these cases were not fatal and the patients made functional recoveries. Heretofore it was believed that the heart being so protected in the rib cage would require such a force to injure it that any force of such intensity would rupture the organ. Prior to Warburg's monograph Bright and Beck[10] analyzed 168 cases, in which death occurred in 157 and rupture in 152. From this study one would be inclined to agree with those who believe that severe force is needed to injure the heart and that when injured rupture is the usual result. Bright and Beck disagreed with this conclusion from their own studies and carried out extensive experimental work on animals to show that enormous force is required to rupture the heart. Severe but lesser forces produce all types of cardiac trauma in which recovery is the rule and rupture the exception. Numerous additional clinical reports[2,3,9,11–16] and experimental studies[17–19] have confirmed this opinion. How, then, can we account for this discrepancy between the earlier reports and the more recent studies?

The earlier reports were based mainly on autopsy findings. Now, however, with our increased knowledge of heart disease in general, our added experience and information from surgery of penetrating injuries to the heart and congenital defects and our improved methods of diagnostic technics (e.g., serial cardiograms, ballistograms, kymograms, cardiac catheterizations,[20] oxygen tension studies, circulation time, etc.), we are able to diagnose and treat injuries that in the past would have been missed or proved fatal. I believe it is now fair to assume from our increasing clinical and experimental knowledge that all degrees of cardiac trauma, from the ultramicroscopic to that of rupture, can and do occur.*

There are several ways in which the heart can be injured, namely: (1) actual direct contusion against the chest wall or spine; (2) a tearing force due to the suspensory nature of the heart; (3) concussion, or what one author[21] calls commotion of the heart, producing minute multiple hemorrhages or vagosympathetic responses;[12] (4) pressure changes within the heart due to compression of distant parts, as with injuries to the abdomen, legs and neck, producing ruptures of valves,[22,23] mainly aortic and mitral, papillary musculature or intercardiac septa;[25] (5) hemorrhage into atheromatous plaques reducing lumen of a previously impaired coronary supply;[19] (6) secondary changes from shock or hemorrhage; and (7) stress.[21] Briefly then, the heart can be damaged from either direct or indirect violence.

Accepting the fact that non-penetrating trauma to the heart is a clinical entity, what then determines the extent of the injury? Several factors are immediately brought to mind: (1) the nature of the injury; (2) intensity of the injury; (3) the presence of shock or hemorrhage; (4) the elasticity of the chest wall;[11] (5) the previous state of the patient's health; (6) the previous state of the heart; (7) phase of cardiac cycle during contusion; (8) the tendency toward vagosympathetic imbalance;[12] (9) rapidity with which general supportive measures are instituted; (10) the recognition of the possibility of cardiac trauma with resultant effective medical or surgical measures.

I believe it is necessary to emphasize the importance of repeated over-all physical examination and not to become focused solely upon some obvious presenting major injury. Many if not most of the patients with non-penetrating trauma to the heart have other serious injuries overshadowing the cardiac trauma which may be at first unrecognized or asymptomatic. I would like to suggest that one consider the possibility of cardiac trauma in all cases of severe trauma, whether or not the chest is grossly involved.

The major symptoms and physical signs of moderate to severe cardiac trauma, many or all of which on original examination may be absent, are listed as follows: (1) unconsciousness, (2) cardiac arrhythmias, (3) tachycardia, (4) chest pain, (5) cyanosis, (6) friction rub, (7) tamponade, (8) dyspnea, (9) angina pectoris, or (10) myocardial insufficiency starting within a reasonable time after the accident. Most authors, and particularly Gore,[9] believe that signs and symptoms must develop reasonably soon after the injury for the heart lesion to be honestly assigned to trauma. Warburg[8] and Pollock,[15] on the other hand, had cases that did not manifest symptoms or signs for weeks or even months following injury. I do not mean to minimize the problems involved in the diagnosis of cardiac contusion or to infer that even the majority of the cases are easy to detect. Diagnosis, under the best of conditions, is difficult, especially in older people with or without known pre-existing cardiac disease. This problem is further complicated by the ease with which cardiac neurosis develops after injury to the chest.[16] Litigation-minded individuals, unscrupulous attorneys and the ultraliberal interpretation of some Workmen's Compensation Boards often tax the patience and integrity of our profession.

Treatment depends, of course, upon the nature of the lesion. Where contusion or bruising of the heart is the diagnosis, essentially the same measures as those used for

* Three cases of non-penetrating trauma to the heart were presented; all were in young individuals with previous excellent health and no history of pre-existing heart disease. All were steering wheel injuries to the chest and all patients sustained multiple fractures of the ribs. Extensive and exhaustive medical investigation by military and civilian consultants was carried through in these cases. Briefly: Case I was a twenty-two year old man with traumatic pericarditis. Case II was a twenty-two year old man in whom all signs and symptoms of aortic insufficiency developed within ten days while he was hospitalized for fractured ribs. Case III was a thirty-five year old man with extensive posterior cardiac contusion. These latter cases were reported in detail but permission to publish them has not been received from government authorities.

coronary thrombosis are instituted. Tamponade requires aspiration or surgical drainage. Rupture of the myocardium, if not immediately fatal, may respond to immediate surgical repair as suggested by Beck in 1935.[1] Rupture of the inner layer of the myocardium or the endocardium can produce aneurysms that may subsequently respond to surgery.[23] Valvular rupture, mainly of the aortic and mitral valves, is fair game for cardiac surgeons. I have no doubt that in the near future, if not actually at the present time, several cases of cardiac trauma will become definite surgical emergencies with results comparable to if not better than that now experienced with some congenital defects.

The medicolegal aspects of non-penetrating trauma to the heart are unlimited. I am afraid that when those in the legal profession become more acquainted with this entity, which they are rapidly doing, our previous difficulties with headache, backache and whiplash injuries to the neck will appear relatively simple. It therefore behooves all of us who handle the problem of trauma to consider the possibility of cardiac damage, record an evaluation of the cardiovascular system and obtain diagnostic studies and cardiac consultation whenever indicated. In this way we can best serve and protect the patient, the insurance carrier and ourselves.

SUMMARY

1. Cardiac trauma, in all degrees from ultramicroscopic to rupture, produced by a non-penetrating force, is now an accepted clinical entity.

2. Methods of injury have been outlined and factors determining severity of injury discussed.

3. Difficulty of and caution in diagnosis is stressed.

4. The possibility of surgical repair of certain cardiac injuries opens a new field in cardiac surgery.

5. Medicolegal aspects of this problem require its consideration in all cases of severe trauma.

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DISCUSSION

Roy Cohn (San Francisco, Calif.): Despite the large number of cases of non-penetrating trauma to the thorax which are seen in the emergency services of City Hospitals, the proved cases of

cardiac damage are small in number. Unfortunately, the proved cases usually are fatal.

One case we saw was that of a twenty-two year old man who died of cardiac failure sixty-four days after being struck on the chest wall by the steering wheel in an automobile accident. There was a large tear in the interventricular septum. Such a lesion is theoretically amenable to repair with modern technics. Unfortunately, the gross damage probably is not the cause of the heart failure. Many patients live on with defects of considerably greater extent. There was also considerable disorganization of the cardiac musculature, which was undoubtedly the most significant factor in the cause of the heart failure.

Because of the ramifications of this problem it is important that in all cases of trauma to the thorax we at least consider the possibility of cardiac trauma and note the cardiac data as a baseline, including in this record an electrocardiogram even though its diagnostic value is questionable.

PAUL C. SAMSON (Oakland, Calif.): Dr. Halter's paper is very timely. Those of us who treat injuries must remain constantly aware of the possibility of non-penetrating cardiac trauma. Recognition is of great importance from a strictly medical as well as from a medicolegal viewpoint. I am sufficiently impressed with the serious potentialities of cardiac trauma to order electrocardiograms *routinely* on all patients who have sustained any degree of bluntly induced anterior chest injury. Often it is well to repeat the electrocardiogram after forty-eight to seventy-two hours, since the first examination may not be revealing.

At the end of the war I studied seventy-five patients with cardiac and pericardial injuries who were seen by surgeons of the Second Auxiliary Surgical Group. This was an incidence of 3.3 per cent of the 2,267 patients with thoracic and thoracoabdominal injuries cared for by members of the unit. Of fifty-seven patients who had strictly cardiac wounds, thirty-six were listed as having contusions and lacerations due to blunt injury and/or blast. Of this group, eleven (31 per cent) died as a direct result of the cardiac trauma. Such injury has been severe enough to cause mural thrombi within seventeen hours.

The exact type of cardiac trauma sustained may not be readily apparent, but in the face of symptoms, so well outlined by Dr. Halter, and of abnormal electrocardiogram tracings, it is mandatory that competent "infarctation care" be instituted. It is particularly important that none but absolutely life-saving surgery be undertaken during the first few critical hours or days. Flouting of this precept will, without question, result in unnecessary fatality.

I would agree with Dr. Halter that there will be patients with cardiac trauma who will eventually need surgical correction. I wonder, however, if any but the rare instance will demand early intervention. I believe it is too important to make an exact preoperative diagnosis to consider many of these patients as suitable candidates for emergency surgery.

This is but a minute criticism, however, of Dr. Halter's successful effort to bring this important subject to our attention.

There is also a question about the repair of an interventricular defect due to cardiac trauma. The patient had a stab wound of the heart, upon which Tom O'Neil operated. The stab wound resulted in a low interventricular defect which was proved both by symptoms and catheterization. The defect was palpated by a finger passed through a cardiotomy opening in the right ventricle, and fascia lata, together with a roll of pericardium, was passed through the defect and anchored in a U-shape and then anchored one end in each ventricular wall. The patient made a good recovery from the surgery and the heart size diminished. I am quite sure this case has not been published.

J. KARL POPPE (Portland, Ore.): I would like to report a case of avulsion of the pericardium as a result of indirect trauma. This middle-aged woman was referred to me with a clotted left hemothorax for surgical decortication one month after a severe crushing injury to her left chest by rolling logs. Previous attempts at intercostal drainage by the referring physician had required prompt removal of the tubes due to cardiac irregularities. I was somewhat surprised during the decortication to note the complete absence of any pericardial covering of the left side of the heart. The myocardium was disfigured and edematous, delaying its immediate identification. The possibility of such a pericardial avulsion should make one moderately cautious about too vigorous attempts at enzymatic decortication of clotted hemothoraces in very severe chest injuries.

A Study of Excised Pulmonary Metastatic Malignancies

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Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or the policy of the Veterans Administration.

FOR some years metastatic malignancy involving the lungs has been of considerable interest to those surgeons who deal with diseases of the chest and their treatment. Curiously enough this interest has apparently only recently been shared to any great extent by other members of the medical profession, most importantly surgeons treating malignancies primary in various parts of the body.

For many years the opinion was widely held that if metastasis to the lungs occurred, all was lost and further efforts at treatment were futile. However, while metastasis to the lungs is still not good, it is not always a hopeless affair.

Isolated efforts to treat pulmonary metastatic malignancies were made as early as 1926 when Divis[5] performed a "lung resection" for metastatic sarcoma, primary in the breast thirty-five years previously. In 1930 Torek[19] removed a right lower lobe tumor which proved to be metastatic adenoacanthoma, primary three years previously in the uterus.

In 1933 Lilienthal[12] performed pneumonectomy for sarcoma metastatic from the uterus.

In 1939 Barney and Churchill[2] recorded a subtotal lobectomy for a metastatic nodule from carcinoma of the kidney, removed fifteen months previously. Five months before nephrectomy the lung nodule was the *first* evidence of illness in this patient. Twelve years after lobectomy the patient was still alive without evidence of disease.

In 1942 Carlucci and Schleussner[3] reported pneumonectomy for removal of a lung tumor which proved to be melanoma with involvement of hilar nodes. The question was raised then whether this tumor was primary in the lung.

By this time more of these metastatic pulmonary lesions were being presented for consideration of treatment, and even unusual metastatic tumors of ordinarily very malignant behavior were removed. In 1947 Maier and Taylor[13] reported the case of a woman who was well for over three years following right lower lobectomy in 1943 for chorionepithelioma. She had had a hydatidiform mole in 1939.

Alexander and Haight (1947),[1] Effler and Blades (1948),[6] Seiler, Clagett and McDonald (1950),[16] and Ehrenhaft (1951),[7] reported series of pulmonary resections for metastatic tumors. The most recent report found was that by Kergin[9] in 1954.

The investigation and diagnosis of these pulmonary lesions originally gave rise to the thought that bronchial involvement was quite rare, but all authors of the various reports, as well as the present author, have found that bronchial involvement is not uncommon and frequently will yield the diagnosis by positive bronchoscopic biopsy.

Cough, sputum and hemoptysis may be presenting symptoms, although admittedly most of the pulmonary metastatic lesions are silent.

As early as 1932 Vinson and Martin,[20] and in 1936 Maytum and Vinson[15] reported the bronchoscopic diagnosis of metastatic malignancy. Freedlander and Greenfield[8] reported similarly in 1942, and mentioned hemoptysis particularly as a possible presenting symptom of metastatic malignancy.

King and Castleman[10] reported an 18.3 per cent incidence of bronchial involvement in 109 autopsy specimens of pulmonary metastatic malignancy.

In 1945 Tinney and McDonald[18] re-emphasized the error of believing that pulmonary metastatic lesions never involve the bronchi.

In the present series of thirty-five cases involvement of a bronchus of major or segmental caliber was proved in nine instances. In eight

of these (22.8 per cent) bronchoscopic biopsy was positive. Bronchoscopy was performed in an effort at diagnosis in twenty-one cases. Although we routinely bronchoscoped most patients in previous years, it is presently our practice not to bronchoscope patients with peripheral lesions unless there is some hope of obtaining bronchial secretions for examination for malignant cells.

This present series of thirty-five cases of pulmonary metastatic malignancies include all instances of such that were presented to the author for diagnosis or treatment, or both. This group consists of thirteen private patients and twenty-two patients in the Portland Veterans' Administration Hospital. In reviewing these it was, of course, immediately apparent that the nine patients not operated upon were statistically of no significance, since obviously there must be many more such non-explorable instances of pulmonary metastatic malignancy, and so they were included in this study simply to reflect one man's experience in this field.

The actual incidence of pulmonary metastasis for malignancy of any one type or organ can be furnished only by those men who treat the primary lesion and who follow-up their patients till death with periodic recheck examinations including chest roentgenograms. Mandeville[14] has remarked that pulmonary metastases sometimes manifest themselves from five to fifteen years after proof of existence of primary cancer. Certainly the present series, as well as those of other authors, supports this statement.

Some notion of the incidence of pulmonary metastatic malignancy can be obtained from figures of necropsies. Willis[21] states that if all malignant growths are considered collectively, pulmonary metastases occur in about 30 per cent of fatal cases; and that if the portal area malignancies are excluded, the percentage incidences of pulmonary metastasis exceed those for metastases in all other situations. He reported pulmonary metastasis in 75 per cent each of instances of chorionepithelioma, osteosarcoma and renal carcinoma; 65 per cent of thyroid cancer; 60 per cent of melanoma; 55 per cent of breast cancer; 40 per cent of prostate cancer; 55 per cent of esophagogastric-intestinal cancer; 30 per cent of oral and pharyngeal cancers; 20 per cent each of hepatic and pancreatic malignancies; 15 per cent of uterine malignancy and 10 per cent of ovarian cancer.

Even with careful follow-up of cases of treated cancer, including chest x-rays, significant pulmonary metastasis can be missed. In studying seventy-nine autopsied cases showing pulmonary metastases, all of whom had chest x-rays within three weeks of death and autopsy, Lee[11] found thirty cases in which the pulmonary metastases were not radiographically apparent, even in retrospective review of the x-rays.

Some of the explored cases of the present series demonstrated many more nodules at surgery than were visible on the chest x-rays.

At the present time most surgeons are uniform in their attitude toward these lesions, when found, whether single or multiple, as long as the involvement is unilateral. Fewer surgeons have thus far attempted removal of bilateral lesions.

There is considerable question in some men's minds regarding the advisability of ever treating a pulmonary metastatic lesion if the primary tumor is a melanoma. Although the literature abounds with suggestions that melanoma can be primary in many organs and sites, as reviewed by Stewart, Hay and Varco,[17] most men believe the skin and eye account for well over 90 per cent if, indeed, not 100 per cent. Thus melanoma encountered in the lung should be considered metastatic even if a primary lesion cannot be found. This was the situation in Case 2. My experience with melanoma involving the lung is very discouraging, yet I still believe excision is warranted when possible instead of accepting a defeatist philosophy.

The sources of the thirty-five pulmonary metastatic malignancies seen by the author from January, 1948, through December, 1954, were: melanoma, five; sarcoma of soft tissues, four; carcinoma of rectum and rectosigmoid, three; carcinoma of colon, three; teratocarcinoma of testis, three; carcinoma of stomach, three; carcinoma of breast, two; carcinoma of lung, two; carcinoma of kidney, two; carcinoma of larynx, one; carcinoma of pharynx, one; carcinoma of head of pancreas, one; seminoma of testis, one; carcinoma of floor of mouth, one; carcinosarcoma of parotid, one; leiomyosarcoma of jejunum, one; and one thought primary somewhere in the gastrointestinal tract. (Table 1.)

Case 31 (seminoma) is included for completeness only, since the pulmonary therapy naturally would not be surgical. However, Case 17 demonstrates how the previous presence of a radiosensitive tumor can confuse and delay diagnosis and treatment.

Case 35 is listed as questionably primary in the gastrointestinal tract because the primary lesion was never identified definitely. The biopsies from thoracotomy were reported as metastatic adenocarcinoma of gastrointestinal origin. This is admittedly possibly erroneous.

Of the thirty-five cases seen, nine never came to exploration for different reasons. Either the lesion could be treated better by irradiation, or the patient was non-explorable, or the diagnosis was entirely missed. From a practical standpoint, although Cases 19 and 20 were not diagnosed correctly as to primary tumor and site before autopsy, this made no significant difference, since they were non-explorable. This was not so in Case 27 where both primary and secondary tumors might have been treated surgically except for unfortunate delay in efforts at non-operative diagnosis.

Of the thirty-five pulmonary metastatic malignancies, twenty-six came to surgical exploration and of these twenty-two were resected. Of these twenty-two, nine were private patients and thirteen were VAH patients. Of the four patients only explored, one was a private patient and three were VAH patients.

Of the twenty-two patients with resectable metastatic lesions, twenty had had surgical treatment of the original primary lesion, one had had irradiation treatment of the primary, and one (Case 2) had had something burned off his right ear "years ago." Examination of his ear was negative. Of the four patients who were explorable but not resectable, three had had surgical treatment of the original primary lesion, and in one (Case 35) the primary lesion was never found antemortem and there was no autopsy. The operative biopsy was reported as metastatic adenocarcinoma from gastrointestinal tract. The four unexplored cases had their primary lesions identified at autopsy (Cases 19, 20, 27 and 30).

Twenty-nine operations were performed upon twenty-six patients. Case 21 had two operations in two episodes, involving first one lung and then the other. Case 16 had three lung operations in two episodes, the first involving both lungs and the second involving only one lung. This patient had a still later recurrence extrathoracically which also was extirpated.

There were four explorations with biopsy, seven segmental resections, eight lobectomies and ten pneumonectomies. Segmental resection includes the usual procedure done from hilus outward and also resection of less than a lobe done from the periphery inward, following segmental and subsegmental planes with individual treatment of encountered structures. Diagnostic excision biopsy is not included and so-called wedge resection or resection between clamps is not included or practiced.

A survey was made of the interval between treatment of the primary lesion and that time when the pulmonary metastasis was discovered on deliberate or chance chest x-ray, or made itself clinically evident by producing symptoms which led to diagnosis. Case 2 had to be omitted from this survey since the primary was only suspected and the patient could not remember when he had the "something" burned off his right ear; therefore, no time interval could be established. Case 35 was deleted since the primary lesion was never proved.

It was found that evidence of pulmonary metastasis was obtained within three years after treatment of the primary lesion in about two-thirds of the twenty-six instances of pulmonary metastasis in twenty-four proved primaries. Furthermore it was noted that in nineteen instances this evidence was a positive chest x-ray in an otherwise asymptomatic patient, while in the other seven instances there were clinical symptoms as the first evidence of pulmonary metastasis.

Fifteen of the instances of pulmonary metastasis had surgery within one month or less of the obtaining of evidence of the presence of the lesion, and fourteen of these were resectable. Twenty-three of the twenty-nine thoracotomies were performed within three months and twenty of these were resections.

There has always been discussion whether metastatic malignancies to the lung or elsewhere, presumably blood-borne to their place of colonization, ever metastasize to the regional nodes. In a recent editorial Kergin[9] mentions that such secondary lesions seem less likely than the parent primary tumor to metastasize along lymphatic channels. Eight of the twenty-

TABLE 1

DATA ON THIRTY-FIVE CASES OF PULMONARY METASTATIC MALIGNANCY AS OF DECEMBER, 1954

Case, Sex and Age (yr.)	Primary Lesion	Treatment	Pulmonary Metastasis							Results		
			No. of Lesions Uni- and Bilateral	When and How Evident after Treatment of Primary	Bronchial Involvement	Bronchoscopy(?), Biopsy(?)	When Explored after Treatment of Primary	Lung Operation	Specimen, Diagnosis and Nodes(?)	Recurrence after Lung Operation	Death after Lung Operation	Alive without Evidence of Recurrence
1. F, 25	Melanoma, right arm	1945, excision; 1948, axillary node dissection; 10/51, ovaries and appendix removed; 1/52, mass, right flank, excised	1 coin lesion, left lung	10/51, check chest x-ray (6 yr.)*	No	No	3/8/52 (6–7 yr.)	Left pneumonectomy	Metastatic melanoma; multiple tiny nodules; all nodes positive	Abdominal mass, 7/52 (4 mo.)	3/53	.....
2. M, 67	Melanoma, site(?)	"Something burned off right ear years ago"(?); exam. of ear negative	1 coin lesion, R.U.L.	3/52, survey x-ray pickup(?)	No	Negative	5/14/52(?)	Right upper lobectomy	Melanoma; coin lesion; nodes negative	"Stroke"; metastasis(?) or CVA(?) 8/14/52 (3 mo.)	10/19/52, no autopsy	.....
3. M, 42	"Synovioma," right foot; later changed to melanoma	9/47, right foot amputation; 10/50 excision recurrence in right thigh	Mass, R.L.L.	3/53, check x-ray (66 mo.)	No	Negative	6/2/53 (69 mo.)	Right exploratory thoracotomy; biopsy	Multiple nodules; metastatic melanoma; nodes positive	Generalized (3–6 mo.)	11/9/53, autopsy, melanoma, general metastases	.....
4. M, 36	Melanoma, right occipital scalp	11/51, junctional nevus excised; 11/52, radical excision, scalp, with radical neck dissection	Mass, R.L.L.	11/2/54, check x-ray (24 mo.)	No	No	11/16/54 (24+ mo.)	Bilobectomy	Metastatic melanoma; nodes negative	.....	.....	As of 12/54 (1 mo.)
5. M, 24	"Fibrosarcoma," left forearm, later changed to melanoma	1/49, incised; 1/50, biopsy; 3/50, mid-humeral amputation	Multiple nodules bilaterally	5/51, check x-ray (14 mo.)	No	Negative	6/21/51 (15 mo.)	Left exploratory thoracotomy; biopsy	Multiple nodules; metastatic melanoma; nodes positive	.....	10/15/51 (bilateral multiple excisions of "fibrosarcoma" had been planned)	.....
6. M, 26	Fibrosarcoma, right thigh	7/18/45, excision	6 nodules, right lung	8/48, check x-ray (36 mo.)	No	No	8/24/48 (37 mo.)	Right pneumonectomy	6 nodules; metastatic fibrosarcoma; nodes negative	.....	.....	As of 12/54 (76 mo.)
7. F, 79	Undifferentiated sarcoma, right thigh	7/49, excision	Mass, L.U.L.	5/54, weight loss, malaise, chest x-ray (58 mo.)	No	No	5/29/54 (58 mo.)	L.U. lobectomy	Metastatic undifferentiated sarcoma; nodes negative	.....	.....	As of 12/54 (7 mo.)
8. M, 20	Angiosarcoma, right leg	7/49, mid-thigh amputation	Mass, L.L.L.	12/13/49, check x-ray (5 mo.)	No	No	1/10/50 (6 mo.)	Left pneumonectomy	7 nodules; metastatic angiosarcoma; nodes negative	Right lung; check x-ray 4/18/50 (3 mo.)	5/23/51	.....
9. M, 29	"Fibrosarcoma," interscapular region	5/45, excision; 10/46, excision of recurrence	Multiple bilateral	2/50, check x-ray (57 mo.)	No	Negative	11/29/51 (78 mo.)	Left exploratory thoracotomy; biopsy	Hundreds of nodules; metastatic "fibrosarcoma"; nodes negative	.....	5/28/52, autopsy, neurofibrosarcoma (After thoracotomy patient	.....

											had right chest opened elsewhere, with idea of removal of nodules, and then left pneumonectomy, but same findings on right)	
10. F, 65	Carcinoma, rectum	11/27/49, abdominoperineal resection	Obstructing L.U.L.	Simultaneously; "pneumonia"	Yes	Yes; metastatic adenocarcinoma	1/8/50 (5 wk.)*	Left pneumonectomy	Metastatic adenocarcinoma; nodes negative	Abdomen (3–6 mo.)	6/17/50, good palliation	.....
11. M, 39	Carcinoma, rectosigmoid	6/48, abdominoperineal resection	Obstructing right intermediate	4/50, cough, etc. (22 mo.)	Yes	Yes; metastatic adenocarcinoma	8/5/50 (26 mo.)	Right pneumonectomy	Metastatic adenocarcinoma, nodes positive	Left shoulder, 8/25/50 (20 days)	12/50	.....
12. F, 75	Carcinoma, rectosigmoid	2/23/48, anterior resection	Mass, L.L.L.	7/50, survey x-ray (29 mo.)	No	No	Recurrence also in bowel	None	.....	.....	11/9/51	.....
13. M, 51	Carcinoma, cecum	7/13/49, resection	Obstructing R.M.L.	6/54, hemoptysis (59 mo.)	Yes	Yes; metastatic adenocarcinoma	9/28/54 (62 mo.)	Right pneumonectomy	Metastatic adenocarcinoma; nodes positive	.....	.....	As of 12/54 (3 mo.)
14. F, 55	Carcinoma, left colon	1/50, resection	1 coin lesion, R.U.L.	7/52, cough, hemoptysis (30 mo.)	No	Negative	10/20/52 (33 mo.)	R.U. lobectomy	Metastatic adenocarcinoma; nodes negative	Brain, 3/14/53 (5 mo.)	5/6/53	.....
15. F, 52	Carcinoma, sigmoid	7/51, resection	Mass, L.L.L.	3/54, cough, sputum (32 mo.)	Yes	Yes; metastatic adenocarcinoma	11/8/54 (40 mo.)	L.L. lobectomy; transdiaphragmatic exploratory laparotomy ("second feel")	Metastatic adenocarcinoma; nodes negative	Nodule(?) right lobe liver(?) ("second feel")	.....	As of 12/54 (2 mo.)
16. M, 28	Teratocarcinoma, left testis	7/51, left orchiectomy 8/51, left retroperitoneal node dissection; 12/51, mass, right groin, excised	2 masses right lung; 1 mass left lung	5/53, check x-ray (22 mo.)	No	No	6/9/53 (23 mo.)	Multiple segmental resection, right	Metastatic teratocarcinoma; nodes negative	Check chest x-ray; L.L.L. mass 11/53 (6 mo.)	.....	As of 12/54 with negative intravenous pyelogram (5 mo.)
							6/25/53 (23 mo.)	Multiple segmental resection, left	Metastatic teratocarcinoma; nodes negative			
			Mass, L.L.L.	11/53, check x-ray (28 mo.)	No	No	12/8/53 (29 mo.)	Segmental resection L.L.L. with panel chest wall	Metastatic teratocarcinoma; nodes negative	Left kidney 7/54 (8 mo.)	.....	
							(7/26/54)	L. nephrectomy and adrenalectomy	Metastatic teratocarcinoma; nodes negative	.....	.....	
17. M, 30	Seminoma, right testis; teratocarcinoma, left testis	10/49, right orchiectomy and x-ray treatment; 12/51, left orchiectomy; 1/52, left retroperitoneal node dissection (negative)	Mass, L.U.L.	5/52, check x-ray (5 mo.); failed to respond to irradiation	No	Negative	10/2/52 (10 mo.)	L.U. lobectomy and subsegmental L.L.L.	Metastatic embryonal carcinoma; nodes negative	Right chest wall, 1/7/53 (3 mo.)	2/17/53	.....
18. M, 28	Teratocarcinoma, left testis	5/10/52, left orchiectomy; 6/11/52, left retroperitoneal node dissection (negative)	Nodule, R.L.L.	11/52, check x-ray (6 mo.)	No	No	11/20/52 (6 mo.)	R.L. lobectomy	Metastatic embryonal carcinoma; nodes negative	.....	.....	As of 12/54 (25 mo.)

TABLE I (*Continued*)

Case, Sex and Age (yr.)	Primary Lesion	Treatment	Pulmonary Metastasis							Results		
			No. of Lesions Uni- and Bilateral	When and How Evident after Treatment of Primary	Bronchial Involvement	Bronchoscopy(?), Biopsy(?)	When Explored after Treatment of Primary	Lung Operation	Specimen, Diagnosis and Nodes(?)	Recurrence after Lung Operation	Death after Lung Operation	Alive without Evidence of Recurrence
19. M, 54	Carcinoma, stomach (autopsy)	Diagnosis missed	Obstructing R.U.L.	Primary never symptomatic or treated	Yes	Yes; "epidermoid" carcinoma, R.U.L.	Positive supraclavicular node	.....	Metastatic carcinoma in node	.....	9/22/50, autopsy, carcinoma of stomach with metastasis to lung	.....
20. M, 55	Carcinoma, stomach (autopsy)	Diagnosis missed	Obstructing L.U.L.	Vague symptoms but primary never diagnosed	Yes	4 times; "epidermoid" carcinoma on 4th bronchoscopy	Extreme pulmonary emphysema	.....	.....	.....	10/29/51, autopsy, carcinoma of stomach with metastasis to lung	.....
21. M, 58	Carcinoma, stomach (cardia)	7/49, resection	Mass, L.L.L.	4/52, check x-ray (33 mo.)*	No	No	4/9/53 (45 mo.)	L.L. lobetomy; excision of strictured anastomosis and reanastomosis	Metastatic adenocarcinoma; nodes negative	Mass R.L.L.; check x-ray 8/54 (16 mo.)	.....	As of 12/54, negative chest and gastrointestinal x-rays
			Mass, R.L.L.	8/54, check x-ray (61 mo.)	No	No	9/7/54 (62 mo.)	Segmental resection, R.L.L.	Metastatic adenocarcinoma; nodes negative	.....	.....	.....
22. F, 60	Carcinoma, right breast	8/50, radical mastectomy	Mass, R.U.L.	3/54, survey x-ray (43 mo.)	No	No	4/21/54 (44 mo.)	Right pneumonectomy	Metastatic carcinoma; all nodes positive	Spontaneous(?) rupture distal third esophagus sometime within early p.o. period	5/15/54	.....
23. F, 58	Carcinoma, right breast	1945, radical mastectomy	Multiple, left lung and chest wall	1/50	No	No	Multiple other metastases	Neck node biopsy	Metastatic carcinoma	.....	11/50	.....
24. M, 58	Carcinoma, left lung	11/18/52, pneumonectomy, left	Mass, R.U.L.	11/53, check x-ray (12 mo.)	Yes	Yes; positive cells	1/19/54 (14 mo.)	Segmental resection, R.U.L.	Metastatic epidermoid carcinoma; nodes negative	Mass R.L.L.; check x-ray 8/54 (7 mo.); clinically well 11/16/54	.....	.....
25. M, 63	Carcinoma, right lung, R.L.L.	1/10/52, R.L. lobectomy	Lingular infiltration	5/52 check x-ray (4 mo.)	No	Negative	6/27/52 (5 mo.)	Segmental resection, L.U.L.	Metastatic carcinoma; nodes negative	3–6 mo., generalized	11/15/52	.....
26. M, 67	Carcinoma, right kidney	4/11/46, nephrectomy	Mass, R.M.L.	6/53, check x-ray (86 mo.)	Yes	Yes; metastatic adenocarcinoma	7/28/53 (87 mo.)	Right pneumonectomy	Metastatic carcinoma; nodes negative; silicosis	.....	7/28/53, left pulmonary edema and silicosis (autopsy)	.....
27. M, 62	Carcinoma, left kidney (autopsy)	Diagnosis missed	Mass, L.L.L., 10/51; bilateral masses, 1/52	Primary not diagnosed although suspected	No	Negative	Thoracotomy on left recommended unless kidney to be explored; bilateral involvement evident 1/52	.....	.....	.....	8/2/52, autopsy, left renal carcinoma with bilateral lung metastases	.....

28. M, 61	Carcinoma, larynx	9/51, irradiation and tracheostomy	Mass, right; masses, both	3/52, check x-ray; 2/53, check x-ray	No	Negative	.....	.....	.....	.....	5/54	.....
29. M, 57	Carcinoma, pharynx	3,4/53, irradiation	Nodule, left lung	8/53, check x-ray (16 mo.)*	No	Negative	9/3/53 (17 mo.)	Left pneumonectomy	Metastatic epidermoid carcinoma; nodes negative	Check x-ray, mass R. L.L. 3/54; larger on x-ray 12/54 but patient clinically well	.....	.....
30. M, 78	Carcinoma, head of pancreas (autopsy)	Not treated; diagnosis suspected	Infiltration, L.U.L.	Symptoms in fall, 1950; chest x-ray	Yes	Negative	Recommended if abdominal workup negative, but exploratory laparotomy planned after x-rays showed duodenal deformity	.....	.....	.....	3/3/51, autopsy, carcinoma of head of pancreas with metastasis to lung	.....
31. M, 59	Seminoma, right testis	1/11/50, orchiectomy and irradiation	Mass, L.U.L.	4/51, check x-ray (15 mo.)	No	Negative	5/51, irradiation to left lung with clearing	.....	.....	.....	.....	.....
			Mass, R.U.L.	12/52, check x-ray	Yes	Yes; epidermoid carcinoma	12/52, irradiation to right lung with failure to clear	Pulmonary emphysema	.....	.....	11/53, autopsy, R.U.L. bronchogenic carcinoma; and metastatic seminoma to right kidney	.....
32. M, 47	Carcinoma, floor of mouth (with neck mass)	6/53, irradiation to mouth, only (?)	Multiple bilateral	5/30/54, "chest cold"; chest x-ray (12 mo.)	No(?)	No	Seen 7/27/54	None	.....	.....	8/54	.....
33. M, 39	Carcinosarcoma, right parotid	8/48, excision; 12/48, radical neck dissection	Multiple masses, left lung	5/53, arthropathy (57 mo.)	No	No	8/26/53 (60+ mo.)	Left pneumonectomy and panel chest wall	Metastatic sarcoma from carcinosarcoma; nodes negative	10/17/53 (2 mo.) neck nodes and right chest wall	11/12/53	.....
34. M, 39	Leiomyosarcoma, jejunum	10/10/52, resection; but in 1948 and 4/52 neck masses had been removed and diagnosed "fibrosarcoma"	Mass, L.U.L.	6/53, check x-ray (8 mo.)	No	No	6/17/53 (8 mo.)	Segmental resection, L.U.L., and removal mass of anterior mediastinal nodes	Metastatic leiomyosarcoma; node mass positive	One leg and 2 jejunal masses removed 9/53 (3 mo.); alive with multiple bone metastases	.....	.....
35. F, 56	Gastrointestinal tract(?), carcinoma(?)	Never diagnosed or proved	Multiple right lung masses	Primary never known; flu and chest pain 8/51	No	Negative	11/26/51	Right exploratory thoracotomy; biopsies	Metastatic adenocarcinoma of gastrointestinal origin on biopsy, nodes all involved	.....	2/52, no autopsy	.....

* Numbers in parentheses indicate lengths of time intervals.

six cases explored had positive nodes. Of course the point could immediately be argued that such nodes were involved from the primary rather than from the pulmonary colony. Nonetheless the practical point is that nodal involvement may be present in about one-third of the instances of pulmonary metastasis. Three of these were in non-resectable cases, of which two were melanomas and one the metastatic adenocarcinoma from somewhere in the gastrointestinal tract. The other five were in resectable cases, two in instances of metastatic large bowel carcinoma, one melanoma, one metastatic breast carcinoma, and one metastatic jejunal leiomyosarcoma.

Of the twenty-six patients explored there were two surgical deaths (7.7 per cent). One of these (Case 26) died about ten hours after surgery, and autopsy demonstrated pulmonary edema in the remaining lung. Whether the silicosis also present contributed to the development of the edema or whether the edema was entirely iatrogenic is not known. The other surgical death was in a sixty year old woman (Case 22) in whom right pneumonectomy was performed for metastatic breast carcinoma forty-four months after radical mastectomy. She did well for six days but then complained of nausea, felt warm and had a temperature of 99.8°F. A right chest tap done on suspicion revealed foul-smelling fatty fluid, and thoracotomy disclosed a 6 inch linear tear in the distal esophagus. She died eight days later and autopsy was not performed. No explanation or clinical event could be found to explain this extraordinary complication which proved to be fatal. The esophagus was not gangrenous since it bled readily when sutured, which was done several times. Although jejunostomy was performed, it was believed she could not tolerate mobilization of the stomach and a thoracic esophagogastrostomy until she was in better condition and the empyema less foul and acute. The situation might have been better handled with an initial closure of the cardia at the diaphragm via the chest, and a sidetracking cervical esophagostomy, with or without immediate resection of the intervening esophagus.

As of December, 1954, eight of the twenty-two patients resected were alive without evidence of recurrence. Three others were alive with evidence of recurrence. One of these is a patient with metastatic bronchogenic carcinoma (primary in the other removed lung), in whom recurrence developed within one year of removal of his pulmonary metastasis. Multiple bilateral pulmonary metastases developed in another patient within six months after removal of pulmonary mtastatic pharyngeal carcinoma. The third patient, whose original primary was leiomyosarcoma of the jejunum, developed recurrence within three months of his lung surgery.

Of five patients seen with melanoma, only three were resectable and only one is alive, and he but one month, as of December, 1954. Three of the four patients with sarcoma of soft tissues were resectable and two are alive, one for over six years (Cases 6 and 7).*

Five of the six cases of large bowel malignancies metastatic to the lung were resectable. This resectability is also the experience of others and in view of the large incidence of colon and rectum malignancies, this would seem to be a worth while group of primary malignancies to search and check very closely. However, only two of the five patients are alive, both without evidence of recurrence, for three months and two months, respectively. Case 15 had transdiaphragmatic laparotomy at the time of left lower lobectomy for a "second feel" and a partial "second look." A nodularity was felt far over and deep in the right lobe of the liver which may prove to have been a contemporary of the metastasis in the left lower lobe. The other three are dead, evidence of recurrence having developed within one, two and two months of pulmonary resection, respectively.

The testicular tumor group seems another that is worth checking very carefully. All three cases seen were resectable, and two of these patients were alive after twenty-five and five months, respectively, without evidence of recurrence. However, Case 16 had three pulmonary resections in two episodes and then had recurrence in the kidney and adrenal which was removed.

The only metastatic gastric carcinoma (Case 21) has previously been reported in part by Chauncey[4] who resected this carcinoma of the cardia in 1949. The author removed the first recurrence and a strictured anastomosis in 1953, and the second recurrence (in other lung) in September, 1954. As of December, 1954, the patient is alive four months without further evidence of recurrence.

* See Addendum, page 252.

SUMMARY

The lessons of this study re-emphasize well known principles of follow-up familiar to us all. Constantly repeated resurvey and recheck of patients treated for malignant neoplasms is necessary. Recurrences may occur long after the primary lesion has been treated. Pulmonary metastasis may give symptomatic evidence in some instances but is best detected by periodic recheck chest x-ray.

Once any abnormality of the lungs is evident in a patient previously treated for malignant disease, it must be assumed to be the result of metastatic involvement until proved otherwise, and, unless definite contraindication exists, prompt exploration for diagnosis is imperative.

The surgical treatment of involvement of the lung by metastatic melanoma is discouraging, but until some other treatment is evolved the author believes resection should be performed, unless other extrapulmonary lesions are also present.

In addition to the aspect of treatment of spread of the neoplasm, removal of pulmonary metastatic malignancies has another worth while aspect. The patients usually feel better, even if they eventually die of their disease. This is especially true when the pulmonary metastasis is endobronchial with resultant obstructive pneumonitis, fever, chills, cough, sputum, hemoptysis and malaise.

The palliative effect of surgical removal of metastases to the lungs is therefore a very worth while feature, and should be strongly emphasized.

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DISCUSSION

Emile F. Holman (San Francisco, Calif.): This is obviously a very instructive presentation. I would like to add just a few experiences which point up the lesson that we should never give into carcinoma until the last breath is drawn.

A young fellow of fifteen had a sarcoma of the t a removed by Dr. King. A year later he presented himself with a story of increasing lassitude and some loss of weight. We found a metastatic mass in the lower lung. We succumbed to the seductive, but often disappointing, blandishments of the radiologist who treated this tumor for a time. It seemed to disappear, but within six months it was back and the patient also had symptoms of cerebral metastases. A year later he was dead.

X-ray therapy is of little importance except in the case of sarcoma of the testis. We have one patient who had a large tumor removed from his

abdomen which proved to be carcinoma of the testis, although there were no testicular abnormalities. He subsequently came to us with three huge "cannon balls" in the chest; these measured about 6 to 10 cm. in diameter. The patient was treated by x-ray and is living three years after the event. In that instance the radiologist had the better of the bargain.

The next patient, a middle-aged woman, had an operation performed for some pelvic disorder and a tumor of the ovary was found which was considered to be benign. She subsequently came to us, having had an x-ray for employment purposes which revealed a small rounded density in the lung. Some lesions in the chest are unrelated to the original difficulty. At operation this mass was found to be adenocarcinoma, probably of ovarian origin. When the slides were reviewed, adenocarcinoma was found in the ovary originally removed. However, this patient is well now two years later.

Some masses revealed by x-ray may appear like metastatic lesions; that is, with sharp borders. However, when we take laminagrams we find that there are little extensions running out from the lesions, suggesting that it is a primary tumor of the lung and not a metastatic tumor. Hence laminagrams may help in differentiating the two lesions.

In another instance, a forty-five year old man presented himself with accidentally discovered masses in the lung. There were three masses, each about 3 cm. in diameter, in various sections of his left lung. Of course, everybody said, "These are metastatic lesions; they look like metastatic lesions." However, there was no evidence of a diseased organ anywhere else. These three masses were removed at pneumonectomy and found to be simple fibromas, probably on the basis of a tuberculous granuloma, having had their inception at a very young age. At any rate, they were hard and contained some calcium which we could not see in the x-rays, but which we could see in the microscopic sections.

Hence one can never be sure of the exact nature of a lesion in the lung, and one should not dismiss a patient on the basis of an inoperable metastatic tumor. One should know what these lesions are, and to know one must remove them more frequently than in the past. Even though you suspect they are metastatic lesions, if they appear to be solitary lesions, one should certainly remove them.

JOHN C. JONES (Los Angeles, Calif.): Dr. Higginson's experience has been similar to ours. I have performed eleven resections upon ten patients for metastatic pulmonary lesions and, interestingly enough, the oldest and earliest patient in the series is still alive and well. However, the rest of the series is not as good.

The first patient with carcinoma of the breast had a right lower lobectomy three years after her breast resection, and she is alive six years later.

The second patient had bilateral disease, i.e., large multiple "cannon balls" in both lower lobes. We did not know the source of the primary lesion, although we were sure that this was metastatic. After resecting the right lower lobe, we found that it was a melanoma. We questioned the patient and found that eight or nine years previously she had had a pigmented mole scraped and treated with acid. There was no evidence of the mole other than a very small scar on her right upper eyelid. We resected the contralateral lower lobe. The patient died fifteen months after her first lobectomy (seven months after the second lobectomy) from melanoma of the liver. In retrospect, while we were in the right chest, I think we should have opened her right diaphragm and observed the liver. We probably then would not have proceeded with the left lower lobectomy, although the patient was considerably improved after the first one and she had come to us about a year and a half after receiving x-ray treatment for the pulmonary lesion.

Another patient had undergone colon resection five years previously; we performed pneumonectomy for metastases and the patient died seven months later.

The fourth patient had rectal carcinoma with a lung lesion five years after resection; this patient is lost and is presumably dead.

The fifth patient had a right lower lobectomy at the age of six for a metastatic tumor from a Wilms's tumor some nine months after his renal resection. He died seven months later.

A pulmonary lesion developed in the next patient eighteen months following rectal resection for carcinoma of the rectum. Death occurred six months after lobectomy.

Another patient had carcinoma of the breast which had been resected in 1939. At that time the surgeon was forced to remove a portion of the chest wall, after which the wound opened and an open, sucking wound with empyema ensued; the patient was first seen by me at that time. She returned twenty-two months ago with a pulmonary lesion. Resection was performed when she was seventy-two. She is now well sixteen years following the original operation. This patient had scirrhous carcinoma of the breast and a well localized pulmonary lesion that could be resected with lobectomy.

The next patient had a breast removed fifteen months before lobectomy and died nine months later.

Another patient who had had resection for carcinoma of the kidney six years before died seven months after lobectomy for metastasis.

The tenth patient had an indefinite metastatic

lesion of lung and is now alive one year after pulmonary resection; however he now has brain metastases.

As Dr. Holman said, you just do not know which one of these patients is going to get a good result from resection of metastatic pulmonary malignancies. I suppose we will just have to keep plugging along hoping that we can help these patients. This is not a very bright or happy picture.

J. Karl Poppe (Portland, Ore.): Another phase of this problem of supposedly metastatic pulmonary malignancies could be mentioned, that is, primary bronchiogenic carcinomas occurring in patients who have had malignancies treated elsewhere in the body. I have seen four patients in the past ten years with primary lung cancers who had had previous resections of the large bowel for primary malignancies. Three of these four patients had been allowed to reach an inoperable stage on the assumption that the lung tumor, which had been followed by serial chest films, was metastatic from the rectum. A more optimistic attitude about resecting metastatic pulmonary malignancies would also save a few primary lung cancer patients.

Ian Macdonald (Los Angeles, Calif.): I think most of us are aware of the fact that in the great majority of patients an apparently solitary pulmonary metastasis from any sort of malignant neoplasm means that there is dissemination of the disease elsewhere. It also means that at one particular moment of time it happens to show in one demonstrable area by means of x-ray studies, which are simply a study of shadows. Unlike Dr. Holman's implications and Dr. Higginson's more guarded inference, I think it would be desirable to wait for a while. No patient of mine with apparently solitary pulmonary metastasis is going to have a "fire-engine" exploration. I would like to have that patient under observation and a recheck study done in three months. At the end of that three months if the only lesion demonstrable is that solitary pulmonary focus, then I would be willing to believe that this may be a biologically dilatory process and that perhaps this demonstrable focus does indeed represent the only area of metastasis and is an erratic, peculiar and bizarre manifestation, speaking in general terms. There are a few such neoplasms, as have been indicated here, such as soft tissue sarcomas and some carcinomas of the colon. But I think that demonstration of a solitary focus in a woman with mammary carcinoma should make us wonder what the situation is when a woman with established cancer of the breast has a supraclavicular node which prompts us to do a biopsy study. To me that is an exact demonstration of incurability; and if it is in the lung, solitary focus or not, it means the same thing. Dr. Holman's demonstration of the metastatic sarcoma (incidentally, I do not believe that Dr. Holman succumbs to the blandishments of anyone, radiologic personnel or otherwise), the mere fact that that lesion showed some rather dramatic regression on radiotherapy would, to me, simply be evidence of the incurability of the situation. I would be grateful to the radiologist for keeping me out of the chest. The fact that a few months later there were extensive metastases was simply proof of the biologic nature of the process.

William Lister Rogers (San Francisco, Calif.): There are certain tumors which are slow in their growth and slow in the growth of their metastases. I would like to mention one or two instances. One of the melanomas which arise in the eyes is very slow to metastasize and generally metastases will appear at a late date—ten to twelve years. We had a middle-aged woman who showed her first metastasis in the left lung twelve years after enucleation, and the metastasis was a solitary one. Excision was recommended as the doctor thought that it was probably a metastatic lesion from the melanoma. However, the patient declined operation. The lesion was observed for four years and during that period of time two additional lesions appeared in the left lung field. On exploration five lesions were removed by simple local excision. Two were found that had not been apparent in the x-ray films. The patient has been well for one year since her operation. I think we should carefully evaluate the merits of each case. I question the desirability of performing pneumonectomy in many of these patients. Certainly I question very strongly performing pneumonectomy in the presence of palpable glands. In my experience removing pulmonary metastatic lesions following mammary cancer has been disappointing.

Joseph A. Weinberg (Long Beach, Calif.): When a surgeon decides that an operation should not be performed because of the occurrence of pulmonary metastases from a distant site, he assumes a tremendous responsibility. In effect, he says that no attempt should be made to prolong the patient's life because such lesions are evidence of a hopeless situation. As Dr. Holman has so well expressed it, efforts should be made to rid the body of cancer up to the last breath of the patient as long as there is hope of prolonging life without discomfort or unsightly deformity. This attitude applies particularly to pulmonary metastases without evidence of metastases elsewhere. A patient who is now under observation in our clinic is an example in point. This man had an amputation of the right lower extremity with hemipelvectomy some months ago for the treatment of osteosarcoma of the femur. A few months later x-ray examination disclosed four metastatic nodules in the right lung and one in the left. Because no new nodules appeared during the next month of observation by x-ray, the lesions

in the right lung were excised locally and the lesion in the left lung is to be excised after a suitable interval. These extreme measures may strike some as being too radical. However, to do nothing is to make certain of death by malignancy; and since pulmonary metastases do not have the same tendency to spread as does the primary lesion, and since even repeated thoracotomies may be performed with little disability or discomfort to the patient, it would hardly be logical for the surgeon to perform as much as hemipelvectomy and then decide against resection of pulmonary metastases.

We owe Dr. Higginson our thanks for bringing up this important and controversial subject.

LYMAN A. BREWER, III (Los Angeles, Calif.): There is one point that has not been raised which I thought should be mentioned as an important criterion for operation in metastatic malignancy of the lungs. In some of our earlier cases we were greatly concerned with the discovery of additional pulmonary metastasis following the excision of the only known pulmonary metastasis or metastases. To obviate the difficulty we are relying more and more upon planigrams or laminagrams to detect tiny metastases not visible on the regular films. We strongly recommend the use of this technic on all patients with metastatic pulmonary malignancy, as it will save them from unnecessary operations.

JOHN F. HIGGINSON (closing): I am very happy and pleased with the interest that has been aroused, and I will try briefly to go over the remarks of the discussants.

Dr. Holman's comment that multiple nodules seen on chest x-ray are automatically considered extensive metastasis and that this may be very fallacious is very important to consider. There are unusual things cropping up, and in relation to his case of multiple granulomas, we had a similar case which we decided to diagnose by thoracotomy and biopsy. It looked like multiple metastasis on x-ray, but on direct inspection and microscopic study it looked like some kind of polyarteritis and, of course, as you might suspect, within a few weeks an article appeared in *The American Journal of Pathology* discussing several cases of what they called sclerosing granulomatosis—a most interesting disease, identical histologically with the one that we explored and biopsied. Such possibilities should be considered and these patients should not be given x-ray therapy.

Dr. Jones' patient certainly indicates the use and value of surgical attempts at extirpation and this agrees with Dr. Weinberg's and my own personal philosophies.

Dr. Poppe has remarked that we should consider the possibilities of multiple malignancies in these patients. However, even if it is a second cancer, it should not immediately be considered that all is lost.

We now believe that all these instances of pulmonary metastases should be screened by complete anteroposterior laminagraphy of the chest, and laterally also, if you believe it is indicated. As Dr. Brewer has said, laminagrams can help considerably in deciding which cases to operate upon.

Dr. Macdonald has a most curious and interesting mind. His approach to problems is often intriguingly different. It may be that he is right in his attitude on this problem, but for the present I prefer my own philosophy of attacking these metastases and removing them if such is possible.

Dr. Rogers has mentioned melanoma of the eye. It is believed that at least 95 per cent of all melanomas, if not 100 per cent, originate in the skin and eye. As you know there are numerous reports of melanomas apparently primary in other sites and viscera. However, these are seriously questioned. We agree that pneumonectomy should not be the routine treatment for metastatic lesions, unless all of the lung is involved. We usually perform lobectomy unless resection has already been performed on one side or the other, in which case segmental resection or even wide local excision is performed.

Again, I would like to thank all the discussants for their interest and comments.

Addendum: Recently one patient (Case 6) was readmitted with superior vena caval obstruction. Angiocardiography suggested obstruction within the right atrium at the ostium of the superior vena cava (confirmed at surgery, May 17, 1955). Involvement was too extensive to permit resection.

Roentgenographic evidence of two coin lesions in the right lower lobe developed in Case 7, an eighty year old woman. No additional surgery is planned.

Rupture of the Thoracic Trachea and Major Bronchi Following Closed Injury to the Chest

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RARE lesions achieve greater importance to the clinical surgeon if their early recognition permits curative treatment. We believe this to be the nature of rupture of the thoracic trachea or major bronchi. The late recognition of this rare injury is attended by difficulties in management which may endanger life or expose the patient to distressing months and years of arduous therapy.

Rupture of the thoracic trachea or major bronchus following closed injury to the chest is a comparatively rare accident. Scannell[1] was the first to report a case treated by immediate thoracotomy and repair. His patient was a ten year old boy who had been struck by an automobile and who had an uncontrollable tension pneumothorax within half an hour of the injury. A careful review of all cases of traumatic rupture of the bronchus had been made previously, however, by Kinsella and Johnsrud[2] and although these authors presented their difficulties with the late management of the lesion, they suggested that early thoracotomy with definitive repair of the tracheal injury might be the procedure of choice. Stricture of the trachea of major bronchi, atelectasis and infection in the involved lung were the late sequelae of the injury, appearing at variable intervals of a few days to months after the injury.

During the past few years we have had experience with the recognition and management of three cases of traumatic rupture of the trachea or bronchi following closed injury to the chest. The importance of early recognition of the injury, the possibilities of curative therapy by immediate thoracotomy, and the serious difficulties associated with the management of later stages of the injury will be apparent from the case reports which follow.

CASE REPORTS

CASE 1. A twenty-four year old white man was admitted to the surgical service on January 8, 1954. Four days previously he had been in an automobile accident. He had sustained a crushing injury to his right thorax, and had been taken to an emergency hospital where right trochar thoracotomy and tracheotomy had been performed for a tension pneumothorax. He improved following this procedure, and his subcutaneous emphysema began to subside.

Examination on admission revealed a sick-looking young man breathing rapidly. There was considerable subcutaneous emphysema. His right chest moved little. Percussion and auscultation disclosed signs consistent with hemopneumothorax on the right. His temperature was 102° F., white blood cells 11,650 with a hemoglobin of 13.2 gm. Culture of the pleural fluid was negative.

X-rays of the chest revealed hydropneumothorax in the right side without rib fractures. Fractures of the second and third ribs in the mid-axillary line were seen on later films. (Fig. 1.)

On January 29, 1954, massive atelectasis of the right lung developed. Bronchoscopy disclosed granulation tissue in the right main stem bronchus. The bronchus was dilated and the atelectasis cleared. However, the atelectasis rapidly recurred and bronchoscopy with dilatation was repeated on two subsequent occasions. On February 21st, the last occasion, a No. 16 esophageal bougie was passed through the occluded area. Atelectasis again recurred a few days later.

The patient was seen in consultation for this recurrent atelectasis and for the peculiar nature of the plug in the bronchus seen through the bronchoscope which failed to disclose other than granulation tissue on biopsy. A diagnosis

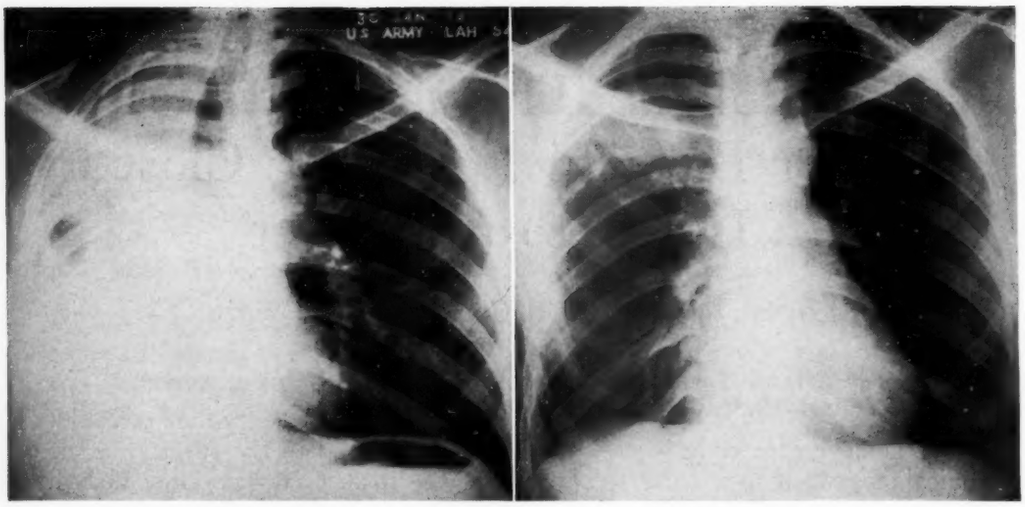

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FIG. 1. Case 1. Preoperative x-ray.

FIG. 2. Case 1. Final x-ray after bronchial reconstruction.

of lacerated right main stem bronchus was made and thoracotomy advised.

On February 25, 1954, right thoracotomy was performed. The chest was entered through the bed of the sixth rib. The lung was atelectatic and covered with fibrinous exudate. The azygos vein was isolated and divided, revealing an inflammatory mass where the right main stem bronchus arose from the trachea. The intratracheal tube was directed into the left main stem bronchus. Further dissection showed the right main stem bronchus to be held to the trachea by a fibrous ring. Apparently the bronchus had been lacerated nearly completely at the original injury and the defect had filled with clot and granulation tissue.

This hourglass area measuring 2 cm. in length was resected and the normal right main stem bronchus was anastomosed to the normal trachea with interrupted silk sutures. The lung was decorticated and expanded fully. The mediastinum was closed. The chest wall was closed with underwater suction drainage.

The patient recovered well and was discharged from the hospital April 12, 1954. He was last seen in August, 1954, and had fully recovered with no residual complaints or pulmonary symptoms. (Fig. 2.)

Comment. The outcome was excellent, but considerable delay in definitive therapy occurred from failure to recognize the true nature of the lesion either clinically or bronchoscopically. An atelectatic lung can regain function on aeration, even if collapsed for several weeks. Fortunately, definitive treatment was carried out early enough so that decortication of the lung could also be accomplished successfully. Previous cases of this type have required pneumonectomy for complete rehabilitation. Early recognition of the nature of the lesion with immediate definitive reparative surgery would have prevented these numerous atelectatic complications. The potential hazards of loss of pulmonary function in one lung would have been averted and total convalescence with complete expansion of both lungs would have been assured in a much shorter time. The ideal treatment for this injury would have been early surgical repair of the laceration of the right main stem bronchus by thoracotomy and bronchial suture.

CASE II. This thirty-four year old white man entered the Stanford Surgical Service three hours after a crushing injury to the right chest. He was in mild shock, breathing with considerable difficulty, and the signs of subcutaneous emphysema over the right chest were rapidly increasing. There were no other injuries. A catheter was placed in the right chest and attached to underwater suction. In spite of this therapy dyspnea and subcutaneous emphysema progressed.

A presumptive diagnosis of ruptured bronchus was made and the patient taken to surgery without further delay.

The right chest was opened through the bed

of the fifth rib. The lung was adherent to the chest and was badly contused. Pneumothorax under some tension was noted on opening the pleura. Just distal to the azygos vein there was a 4 mm. tear in the right main stem bronchus. This was easily closed with interrupted sutures. The patient improved immediately upon restoration of his breathing capacity.

The chest wall was closed with catheter drainage. An uneventful convalescence followed the operation, the right chest was completely expanded and the lung was well in three weeks.

Comment. Early diagnosis with definitive early surgical repair of the ruptured right main stem bronchus prevented complications in the injured lung and resulted in a rapid and complete re-expansion of the lung with maximal preservation of pulmonary function. Convalescence was also markedly shortened by this direct surgical approach.

CASE III. Patient G. H. entered the Stanford University Hospital on August 21, 1954. Early in July, 1954, he had been in an automobile accident, had lacerated his lip, and been knocked unconscious. He regained consciousness and was taken to an emergency hospital where the lip laceration was sutured and he was dismissed ambulatory. That evening he called his family physician because he noted a crackling sensation in his neck. His physician admitted him to the hospital, took x-rays which showed mediastinal air and a fracture of the fifth cervical vertebra with minimal displacement. Over the next ten days the mediastinal and cervical emphysema subsided but the patient noted increasing difficulty in breathing. On the night of the tenth hospital day dyspnea was marked and tracheotomy was performed. No relief was obtained from the dyspnea, and bronchoscopy was performed. A peculiar filling defect was noted in the lower trachea about 2 cm. above the carina. It appeared to be granulation tissue and dilatation of the area was performed. Dyspnea improved, but recurred the next day. Bronchoscopy was repeated and much of the protruding granulation tissue and cartilage was removed with a biopsy forceps. However, a clear airway could not be obtained and a polyethylene tube was inserted through the tracheotomy tube below the area of tracheal disturbance. Over the next six weeks the tube was kept in place and then removed. The following day the patient lost consciousness from anoxia; bronchoscopy was attempted but the bronchoscope could not be passed beyond the area of tracheal obstruction. With difficulty a No. 3 long polyethylene tracheotomy tube was inserted beyond the area of tracheal stenosis, an airway was established, consciousness returned, and the patient was transferred to our care.

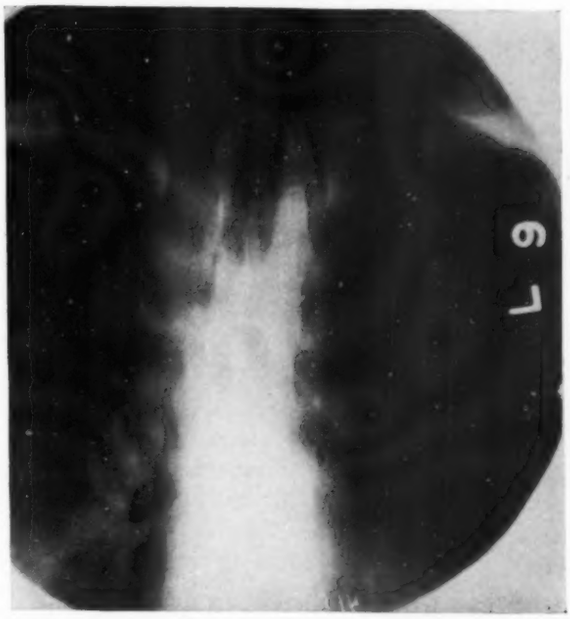

FIG. 3. Case III. Preoperative x-ray showing tracheal defect.

Bronchoscopy disclosed rigid tracheal stenosis about 2 cm. above the carina. The bronchoscope could not be passed through it, nor could the area be dilated. All that could be accomplished was the reinsertion of the smallest polyethylene tracheotomy tube, for asphyxia was pronounced and the airway was completely occluded in its absence.

A diagnosis of traumatic rupture of the trachea with secondary tracheal stenosis was made and surgery advised. Prior to surgical intervention the area of stenosis was outlined with tomography and found to be over 1 cm. long and located within 2 cm. of the carina. (Fig. 3.) We were confronted with the possibility of having to bridge a defect in the trachea once the stenosis was removed.

Thoracotomy was performed through a right posterolateral approach excising the major portion of the fourth rib. The azygos vein was divided, and the right main stem bronchus identified and traced upward to the carina. The entire lower trachea from the thoracic inlet down to the carina was found enveloped

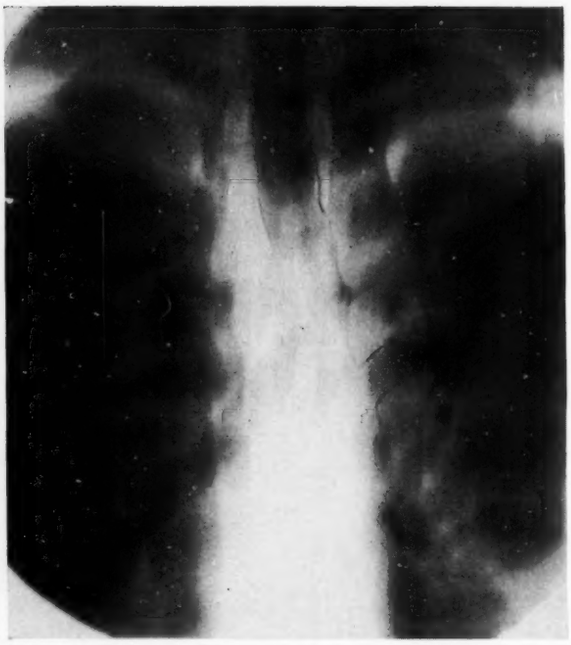

FIG. 4. Case III. Postoperative x-ray with tracheal reconstruction.

in fibrous tissue, and it was dissected free with difficulty from the vena cava, aorta and innominate vessels. An area of stenosis was found starting about 1 cm. above the carina and extending upward for 1 cm. The area of stenosis was incised, and with some difficulty the intratracheal anesthesia tube was finally passed beyond it. We had difficulty in inflating the lungs and finally found only one position of the intratracheal tube that would permit us to continue work. Once satisfactory airway was obtained the area of tracheal stenosis was excised, and with excessive tension it was found possible to pull the edges of the tracheal defect together with double 2 cotton sutures. A No. 8 tracheotomy tube was left through the anastomosis, and the chest closed with underwater seal.

The patient had considerable distress in the postoperative period related to the exact placement of the tracheotomy tube. It had to be removed on the fourth postoperative day. On the sixth postoperative day progressive dyspnea was present. The patient had to be anesthetized and a No. 8 tracheotomy tube was reinserted. Since that time a No. 8 tracheotomy tube has remained in place, being changed first every few days with cauterization of the granulations at the suture line. The tracheal walls have finally healed but removal of the tube is still impossible, and now it is changed once every three weeks. (Fig. 4.) It is apparent on removal of the tube that closure of the airway will occur if it is not replaced. The tube is corked off at the tracheotomy site so that the patient speaks normally and lives a full and active life; however, he cannot go without the plastic airway which still extends down to the carina.

Comment. This case illustrates the profound difficulties resulting from the late occurrence of tracheal stenosis subsequent to injury to the trachea. Initial recognition of the nature of the injury was difficult, but its true nature was apparent at the time of the first bronchoscopy. In retrospect, it would have been advisable to have performed thoracotomy at that time with reconstruction of the trachea. The biting away of the protruding mass in the tracheal lumen resulted in further loss of continuity of the tracheal wall, contributed to the increased extent of the later tracheal stenosis, and heightened the total difficulties of reconstruction. The sequence of events in this case strengthens our conviction that early diagnosis and reparative surgery prior to the development of late sequelae is desirable. Possibly reconstruction of the trachea will again have to be attempted in this patient, but at the moment there seems to be no satisfactory method of bridging a complete defect of this length in the trachea, and we have a sound functional and physiologic result with a tracheal plastic prosthesis. A better anatomic result might never be gained at this late stage, but could have been accomplished by preventive surgery.

MECHANISM OF INJURY

Traumatic rupture of the trachea or main stem bronchus is occasionally due to an associated rib injury or to a penetrating wound of the thorax. Sanger[3] has reported his experiences with this group and has recorded two cases with shell fragment injuries of the right main stem bronchus which were explored early, sutured and repaired with excellent immediate results. In closed injuries to the chest there must be a sudden increased intratracheal or intrabronchial pressure against a closed glottis with a simultaneous regional strain or shear upon the tracheal or bronchial cartilage, bringing about its rupture. The point most likely to tear appears to be the main bronchus at or within a few centimeters of the carina. The tracheal

injuries have been located within 2 cm. of the carina, and the main stem bronchial injuries have also been close to the carina. Rupture of the more delicate lung parenchyma has not been a frequent concomitant of this particular injury.

CLINICAL PICTURE

Early. The initial clinical picture is worthy of emphasis since definitive reparative surgery will be accomplished to the patient's benefit if the disorder is properly recognized. Leakage of air and blood characterize the early symptomatology, and the degree of shock and respiratory distress vary with the amount of air or blood lost. If the trachea ruptures into the mediastinum, mediastinal and suprasternal emphysema appear. If the pleura is also torn, a tension pneumothorax develops. Dyspnea and respiratory distress follow. A progressive uncontrolled tension pneumothorax which will not yield to underwater needle aspiration or even catheter aspiration demands immediate thoracotomy, since this is the cardinal sign of a ruptured trachea or bronchus.

Bleeding occurs in varying amounts. A tear of the bronchus may only result in mild to moderate hemoptysis. This blood may be a contributing factor in the production of atelectasis, and the clot in the trachea or bronchus adherent to the rim of the fractured cartilage forms a scaffolding for ingrowth of granulation tissue which initiates the later tracheal or bronchial stenosis. Occasionally bleeding may be extreme, depending upon the vessel torn; and if one of the major pulmonary vessels is torn into the pleural cavity, a massive hemothorax complicates the tension pneumothorax.

Uncontrollable emphysema or tension pneumothorax after a closed injury to the chest, particularly if accompanied by some degree of pulmonary bleeding, warrants immediate thoracotomy with a tentative diagnosis of traumatic rupture of the trachea or bronchus. The preoperative location of the injury can be determined by bronchoscopy.

Later. If the patient survives the initial injury without the necessity of emergency surgery, respiratory distress follows the tracheal injuries within a period of seven to ten days. Bronchoscopy will usually be performed at this stage of the illness, and it is important that the bronchoscopist recognize the true nature of the tracheal lesion encountered. The granulation tissue membrane protruding into the tracheal lumen represents a displaced segment of tracheal wall with hematoma formation around it and ingrowth of granulation tissue. Extensive biopsy of the lesion to relieve the tracheal stenosis results in an increasing defect in the tracheal wall. Reparative surgery at this stage again seems indicated.

If the lesion has occurred in a main stem bronchus, atelectasis with marked mediastinal shift occurs. Infection is rapidly superimposed upon the atelectatic lung. The cause of the atelectasis should again be clearly recognized by the bronchoscopist. Simple dilatations have proved ineffectual, and reparative surgery of the bronchus is indicated.

Late. Tracheal stenosis with respiratory distress follows continued conservative management of tracheal injury. A malunion of the fractured tracheal cartilage occurs and scarring and stenosis is progressive. Late repair of the trachea becomes necessary. The difficulties of this situation will be discussed later.

Bronchial stenosis with repeated atelectasis and infection of the lung is the aftermath of main stem bronchus injury. Late repair of these lesions is easier than in tracheal stenosis. The very late cases with an atelectatic infected lung require pneumonectomy or lobectomy.

TREATMENT

Kinsella and Johnsrud[2] reviewed all reported cases of traumatic rupture of the bronchus up to 1947, finding thirty-eight in all. Nine of these died within the first hour of injury, and ten more succumbed within the next three days. Tension pneumothorax and bleeding were the prime causes of the early deaths.

Nineteen recoveries were recorded. Ten of these occurred spontaneously from minor injuries which did not require any specific treatment, and the true diagnosis was not made at the time of injury. In the rema ning nine cases varying degrees of stenosis and atelectasis occurred as late sequelae, requiring bronchial dilatations or thoracotomy for improvement.

Early. A tension pneumothorax which cannot be controlled by underwater suction or exsanguinating intrathoracic bleeding after a closed injury to the chest demands bronchoscopy and immediate thoracotomy. Repair of the ruptured trachea or bronchus should result in immediate improvement and rapid recovery

of the patient. Similarly, bleeding from an associated vascular injury can be controlled. Significantly, in the recorded cases ten of the patients who succumbed did survive for a sufficient time to have permitted thoracotomy with possible repair of the injured trachea. Rapid and complete restoration of pulmonary function without late sequelae seems to be the outcome of this early therapy from our own limited experience and from a review of the few recorded cases similarly treated.

Later. The cases of traumatic rupture of the bronchus or trachea not requiring emergency thoracotomy commonly come to bronchoscopy within the first two weeks of injury from respiratory distress, mediastinal shift or atelectasis. Recognition by the bronchoscopist of the nature of the injury at this stage is important, and a review of our own experiences and that of the literature would suggest that definitive surgery should again be accomplished at this time. It is possible that the injuries with minimal cartilaginous displacement could respond to repeated bronchoscopic dilatations, but it seems important to emphasize that the bronchial wall should not be repeatedly biopsied away by the bronchoscopist. It seems more advisable to treat the patients with any significant degree of bronchial displacement or incipient stenosis by open thoracotomy with direct suture of the injured bronchial or tracheal wall. Again this course should prevent troublesome later stenosis, atelectasis and infection. Complete and rapid re-expansion of both lungs is insured.

Late. The problems in the late management of these injuries are extremely difficult and often insurmountable. The bronchial lesions are more amenable to surgical attack, and the involved bronchial stenosis may be resected with end-to-end anastomosis of the bronchial wall. This would be the treatment of choice. However, if infection has been superimposed on the atelectatic lung, lobectomy or pneumonectomy may be necessary.

The most difficult cases are those with late stenosis of the trachea. This stenosis is located close to the carina, and extends over a distance of 1 to 2 cm. Case III represents an example of this type. Prevention of this late sequela is obviously most desirable. The stenosis may yield to repeated bronchoscopic dilatations if very mild, but significant stenosis will not permit an adequate airway between dilatations. Reconstructive surgery of the trachea is difficult, but the following methods should be mentioned:

1. Resection of the stenosis with end-to-end anastomosis. This is the most desirable method. Unfortunately, difficulties in the repair are great because of scarring and fixation of the involved structures. Whereas in animals large gaps in the trachea can be easily overcome, in man the length of the trachea is only 10.5 cm. and it is difficult to gain 2.5 cm. of mobility within the thoracic cavity after the trachea has been injured. This method was attempted and accomplished with questionable success in Case III. A plastic prosthesis is required for weeks to months after satisfactory suture of these late cases.

2. Resection with replacement by a tracheal homograft. The experimental results in tracheal homografting of animals have not been sufficiently encouraging to warrant this technic in man.[4]

3. Resection with replacement by a re-enforced dermal graft. Gebauer[5] has reported excellent success with repair of old tuberculous structures with re-enforced dermal grafts. Experiences with re-enforced dermal grafts of the entire tracheal wall have been too rare to be properly evaluated in man. Rigidity of the graft is essential or a tracheal lumen will not be maintained.

4. Resection with repair with a viable pedicle graft. Penton and Brantigan[6] have reported successful repair of an extensive tracheal defect with a viable pedicle graft formed from a portion of the chest wall consisting of intercostal muscles, periosteum of rib beds, parietal pleura, and intercostal vessels and nerves. An excellent blood and nerve supply can be retained from the intercostal vessels and nerves which enter the pedicle graft posteriorly. No other experiences with repair by this method have been reported so that it is difficult to evaluate.

With the extreme difficulties of late repair of the trachea the importance of prevention of tracheal stenosis from traumatic wounds of the trachea assumes increasing importance.

SUMMARY

Three cases of closed injury to the trachea or bronchus have been reported. These cases emphasize the importance of early recognition of the injury and again illustrate the difficulties

of late repair of tracheal stenosis. Uncontrollable emphysema or tension pneumothorax after a closed injury to the chest warrant bronchoscopy and immediate thoracotomy with a tentative diagnosis of traumatic rupture of the trachea or bronchus. Progressive dyspnea occurring within ten days after closed chest injury demands bronchoscopy. Granulation tissue projecting into the lumen should be recognized as unmistakable evidence of injury to the trachea or bronchus, and thoracotomy with repair of the injury should be accomplished. Otherwise tracheal stenosis will occur later, and the difficulties of late repair are often insurmountable.

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DISCUSSION

William Lister Rogers (San Francisco, Calif.): Drs. Richards and Cohn have had an enviable experience with some of the complications that may arise from non-penetrating injuries to the chest. A direct force or blow may also result in a laceration of the larynx or upper trachea. Such injuries require prompt action. An incision affording adequate exposure of the injured segment, surgical repair and concomitant temporary low tracheotomy together with maintenance of a clear airway will ordinarily afford the desired result.

Similar forces may also produce a tracheoesophageal fistula. Eight such incidents have been reported; the last six were operated upon electively and successfully. One of these six was the result of a blast phenomena recently reported by Capt. Storey.

The authors have properly stressed certain points in management: (1) The early stabilization of the respiratory tract, the necessity of prompt control of escaping air. Emphasis may vary—relief of a tension pneumothorax may suffice in one instance whereas in another suprasternal decompression with possible tracheotomy may be required. (2) The early stabilization of the cardiovascular system. The replacement of blood loss and the release of mediastinal and/or pericardial pressures causing circulatory collapse from increased pressure of the large vessels or heart.

I should like to stress the advisability of using large tubes or catheters for drainage. In these cases needles or small catheters are not apt to be adequate to bring about stabilization.

The value of early bronchoscopy for clarification and localization has been properly stressed. Dr. Hood, who has recently returned from Ann Arbor to Oakland, has reviewed with me three rather similar cases which were treated on the Thoracic Service during a two-year period. All three were stabilized by conservative decompression measures. One with a rent in the membranous trachea, lower third, healed satisfactorily and did not require elective repair. The other two required elective repair, one of the left stem bronchus, the other of the right intermediate bronchus. All end results were satisfactory. I hope these cases will be published later in detail by the Michigan group.

I believe as we gain in experience with this group of tracheal and bronchial injuries the tendency will be toward earlier definitive repair, thus avoiding the probability of late stricture formation.

However, it is my present conviction that a vigorous attempt to obtain prompt cardiorespiratory stabilization by conservative measures is highly desirable. If successful, it should be followed at an early date by bronchoscopy. This sequence of events will still fit admirably into the pattern of early definitive repair.

I wish to thank the authors for the privilege of discussing their stimulating paper.

Paul C. Samson (Oakland, Calif.): I enjoyed this paper very much. I am sure that tears and rents of the major bronchi occur much more frequently than any of us realize. I would advise also that this particular type of injury constitutes one of the few occasions for emergency thoracotomy and early suture of the defect. Those of us who had some control over thoracotomies during the war learned rather rapidly that one could do too many thoracotomies and that in contradistinction to the situation in the abdomen, we were better advised most of the time to stay out of the chest unless exact indications existed. The mere factor of an uncontrollable pressure pneumothorax, however, is sufficient reason for performing thoracotomy. I do not know whether being more a chest surgeon than an abdominal surgeon makes one use bigger tubes or not, but we put in thirty-six to forty tubes instead of sixteen to twenty, so we usually get adequate drainage.

I would like to discuss some of the aspects of the later complications and limit my remarks to the bronchi. There are two types in these cases: The first is an incomplete stenosis of a bronchus, and in these intermittent bouts of distal infection occur. I want to emphasize that the distal infection need not show up as bronchiectasis on the bronchogram, but the history of repeated episodes of so-called pneumonia is sufficient. Such patients must have some type of resection to recover. If the stenosis is lobar, lobectomy will suffice; if it is segmental, segmental resection; and if it is a main stem stenosis, sooner or later pneumonectomy will be necessary.

The second type of injury occurs rather rarely and is very dramatic (Dr. Pinkham referred to it). This is a complete traumatic transection of a bronchus. Those who have not considered the possibility may find it hard to realize that a bronchus can be torn apart completely, each end can seal without tearing vessels, and therefore the patient can survive. I would like to mention three patients with this injury that I have treated.

The first patient was seen several years ago and the x-ray which was taken sometime after the injury showed a completely atelectatic left lung. It was our thought that this patient had an organizing hemothorax, since aspiration revealed a little fluid. Luckily, we performed bronchoscopy prior to the proposed thoracotomy and found a complete bronchial obstruction. The patient had fever and a cough, and we thought there was infection in this lung. Therefore we performed thoracotomy and pneumonectomy later. After tying off arteries and veins, much to my horror, the lung dropped out in my hand. This patient made a perfectly uneventful recovery, but lost a lung. We had assumed, and did for sometime thereafter, that these lungs were destroyed. When opened, the bronchial stump exuded a very thick, tenacious, mucoid material, and the bronchi were dilated. This was written off as total bronchiectasis due to complete bronchial obstruction.

The next patient I will mention only briefly as we made this diagnosis in the Stanford outpatient department. The patient went elsewhere and had relatively unsuccessful surgery.

The third patient was first seen at the age of twelve years when it was thought that he had a totally collapsed lung due to tuberculous stenosis. Last year, at the age of sixteen, a re-examination in the light of the story that at the age of one year he was run over by a wagon wheel and had been dyspneic ever since led us to suspect that possibly this was a complete traumatic resection. Dr. Evans of Fresno bronchoscoped the patient and the bronchial stump proved to be fairly large; the rest of the chest was blanked out. We determined to operate upon this patient. In June, 1953, at thoracotomy we found a right lung about the size and shape of a crêpe suzette that looked to be about a year old, totally atelectatic, of course. The vessels were completely dissected free. The upper bronchial stump was identified easily. The bronchial stump which was attached to the lung was in two segments. This was dissected down, first along the bronchus intermedius and then along the apical segment of the upper lobe and the posterior segment of the lower lobe. We developed a bronchial opening which was about 1 cm. across. Rubbery gelatinous material exuded from the lung. By the use of a No. 8 urethral catheter in each of the segmental bronchi with alternating suction and pressure we got complete expansion of this lung. The upper bronchial stump was then beveled and end-to-end anastomosis was performed. Approximately nine months later bronchogram showed a good alveolar pattern and bronchoscopy by Dr. Evans showed that we had a good suture line without stenosis and a perfectly functioning lung.

As far as I know this is the longest time which has existed between injury and successful repair. It well illustrates the dramatic rescue of a lung which can take place and confirms the experimental studies of Webb and Burford.

In conclusion I would like to tell you of this patient's respiratory function. Unfortunately no preoperative function studies were performed, but he had obvious dyspnea on moderate exertion, climbing stairs, etc., and was never able to engage in sports. One month postoperatively his maximum breathing capacity was 72, his walking ventilation was 20 and his ratio 0.28. It was believed that his right lung had developed approximately 16 per cent of the total function. Twenty months postoperatively his maximum breathing capacity had changed to 90, his walking ventilation was 18.3, his ratio was 0.20 (a much better ratio). His right lung was estimated to maintain 20 to 25 per cent of the function. He is attending high school full time, engages in some sports and participates in the regular physical education program without restriction and without undue exertional dyspnea.

If I remember correctly, there was some question about credit for the first bronchial repair, and I believe Dr. Scannell's name was mentioned. Actually, I have it that Dr. Griffith in England was one of the first to do an early definitive repair on a fractured bronchus. I do not have the paper before me, but should you wish to look it up, the reference is as follows: *Thorax*, 4: 105, 1949.

Byron H. Evans (Los Angeles, Calif.): I am sorry to get up after Dr. Paul Samson's very notable success and present my two failures. The first patient was a thirty-four year old man who was injured in an automobile accident. In November, 1951, he sustained a pneumothorax on the right and was treated by immediate needle drain-

age. I saw the patient on approximately the third postinjury day, at which time he was almost moribund. He was cyanotic and there were numerous rales and rhonchi throughout the left lung. He was totally unable to cough and raise his secretions. His pneumothorax was not controlled by needle drainage and, as a consequence, a catheter drainage was established. The patient was transferred to the Veterans Hospital, and right thoracotomy was performed. As his chest was being opened the patient sustained cardiac arrest. The chest was rapidly opened and the endotracheal tube was found to be protruding from the right main stem bronchus. It was forced back into the trachea, directed into the left side, and the tear was found to be a complete transection of the right stem bronchus, extending into the right upper lobe bronchus. The heart was massaged during this time and was able to resume a normal beat. The bronchial tear was repaired. It was necessary to use a small graft to conserve the integrity of the right upper lobe bronchus. The patient survived the operation, only to die shortly thereafter without gaining consciousness at any time.

The second patient was seen in December, 1954, at which time he was in acute respiratory distress. A tension pneumothorax was thought to exist. The resident placed a catheter into the right chest. Tracheotomy was performed. Bronchial aspiration revealed bloody secretions. The patient showed little improvement and as a consequence he was placed on suction, which only further interfered with respiration. This patient was seen by us approximately seventy-two hours after admission to the hospital, and at that time the oscillation in the tube from the chest drainage showed loss of air on inspiratory and expiratory phases. It was apparent that there was a bronchial tear; the patient was operated upon. Again it was found that almost a total transection of the right stem bronchus was present. Ninety per cent of the bronchus was torn and the tear extended into the upper lobe bronchus and into the lower lobe bronchus as well. This patient had marked respiratory difficulties during the anesthetic and sustained a cardiac arrest from which he could not be recovered.

I wish to present these cases to emphasize that this is a marked emergency and both of these patients might have been repaired satisfactorily if the status of the remaining lung had been such that the patient could be anesthetized and carried through the procedure.

The Differential Diagnosis of Alimentary Tract Obstructions in the Newborn

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Obstructions of the alimentary tract furnish more than 80 per cent of the grave surgical emergencies seen in the newborn. So deadly have they been in the past that in 1932 but nine survivors of atresia of the intestine could be counted in all the literature. The first successful repair of an atresia of the esophagus was accomplished in 1941. Total mid-gut volvulus was poorly understood and the distorted anatomy too often became clear only at postmortem examination; while meconium ileus and neurogenic obstruction were, and still are, attended by an extremely high death rate. Today all of the nation's larger centers report increasing numbers of survivors, due largely to earlier diagnosis, prompt surgical intervention and improved pediatric care. This paper is concerned with early recognition; and while the all-inclusive diagnosis of "obstruction" is of first importance, a more concise differentiation offers many advantages in planning, preparation and surgical approach.

Such a differentiation can usually be made quite accurately. The causes of obstruction are relatively few, and several of these are seen only at a single, but definite, location. Conversely, there are certain anatomic levels at which but one type of lesion can occur. These two related statements will be discussed separately.

CAUSES OF OBSTRUCTION

Atresia is a developmental defect, intrinsic in type, and by definition implies complete imperforation. It may occur in any of the body's hollow tubes, and as applied to the alimentary tract it may be found at any level. It is the only type of obstruction found above the duodenum, or below the sigmoid. For completeness, it may be added that stenosis, in the form of a tiny opening through an otherwise imperforate diaphragm, differs from atresia only in degree; it is less common, and for practical purposes is considered under the same heading.

Mid-gut volvulus consists of a volvulus of the entire small intestine. The afferent loop in the twist is the first part of the jejunum, while the exit loop is some part of the colon. It is made possible through non-rotation of the embryonic mid-gut, this in turn producing lack of posterior mesenteric fixation. The symptoms are those of high jejunal obstruction, and this is the only level at which this entity occurs.

Diaphragmatic hernia may include and obstruct any portion of the alimentary tract below the diaphragm. This condition, under the term "upside down stomach," received wide publicity in the past. Within the first ten days the symptoms are more likely to be cardiorespiratory than intestinal. Intermittent periods of collapse with dyspnea, cyanosis and extremely rapid respirations are suggestive symptoms. The defect is usually in the left diaphragm and auscultation may be misleading. A roentgenogram of the chest is the most valuable diagnostic measure.

In meconium ileus the obstruction is in the lower ileum and is caused by a column of thickened, sticky meconium which is dessicated and contracted in its lowest portion. It is caused by pancreatic insufficiency and is early evidence of cystic fibrosis of the pancreas. The infant may be born with a prominent abdomen which becomes more distended as air is swallowed. The first meconium is absent and the rectal sphincter is small and tight. X-ray findings will be discussed later.

Neurologic obstruction due to myenteric plexus deficiency, or early Hirschsprung's disease, occurs only in the colon—usually the left. Distention and vomiting may be slow in developing. The first meconium may be brownish and thin, rather than sticky and tarry-black. Rectal examination usually shows a firm, resistant and tubular pelvic sigmoid. A single roentgenogram, following the administration of a barium enema under low pressure, furnishes the necessary information. It will demonstrate a somewhat enlarged left colon above a rela-

tively smaller lower segment. This distention does not have to be great to be significant in the newborn, for there is no other type of congenital obstruction which produces an enlarged, or even a normal, left colon. Imperforate anus is of course an exception, but in these a barium enema is obviously impossible.

These five subheads will account for the causative factor in nearly all alimentary tract obstructions within the first ten days of life. Returning now to the various anatomic levels, consideration will be given to the symptoms from obstruction at each.

ANATOMIC LOCATION OF OBSTRUCTION

The highest point is the nasopharyngeal airway, and the obstruction is a choanal atresia. It is not within the realm of the average general surgeon but is mentioned because it might well be called the roof of the alimentary tract since digestive mucous membrane is found on its lower, or posterior surface. It is a partition, either bony, fibrous or membranous, across the nasopharynx and blocks the nasal airway. The normal newborn is instinctively a nose breather and obstruction produces serious distress. He pulls for air against closed lips; he cannot close his mouth during sleep, or over a nipple. The one thing he can, and does do, is cry; this aerates him, but he cannot rest. The diagnosis is usually made with a probe, followed by the instillation of a few drops of opaque oil, and a roentgenogram. Treatment in our hospital is carried out by a nose and throat specialist. Seventeen patients have been seen and treated, and it is interesting indeed to note that one of these was born with both choanal atresia and an imperforate anus. This is really going to extremes.

The next lower level is the upper third of the esophagus—the site of atresia of the esophagus, with or without tracheo-esophageal fistula. No diagnosis is more easily made. The symptoms are a wet mouth, frothy mucus, and dyspnea and coughing on any attempt to swallow. A catheter passed down the throat meets an obstruction at about 12 cm., and the instillation of 1 to 2 cc. of opaque oil followed by a single x-ray film in the upright position makes the diagnosis certain. In cases of fistula, the roentgenogram will also show a proportionately larger amount of air in the stomach than in the intestines. In cases of atresia without fistula, the entire abdomen is opaque.

Continuing downward the next level is the duodenum, either above or below the ampulla. Only atresia can be the causative factor here, and vomiting is the one early symptom. This is an important anatomic location, for bile furnishes the color for the first meconium. If diverted upward by an obstruction below the ampulla, the vomitus is green and the stools are colorless. The reverse is true when the point of obstruction is above the ampulla. The x-ray flat film, in either instance, shows a dilated stomach and duodenum, and no air in the intestine below.

A few inches further down, at the first portion of the jejunum, either of two causative factors may produce obstruction, namely, atresia of the jejunum or mid-gut volvulus. They are easily differentiated. Both produce green vomitus, never brown. Atresia allows only grayish mucus per rectum, while in volvulus some meconium has usually accumulated before the twist occurs. The great aid here is again the roentgenogram. In atresia, dilatation down to the obstruction is seen, then no other air; in volvulus, some little air gets through the twist and enters the closed loop, appearing as small arc-like shadows without haustrations. In any case of malrotation a barium enema will usually show the cecum in the right upper abdominal quadrant, or otherwise displaced from its usual location.

A notable exception to the usual rule regarding green emesis may be mentioned here. Certain cerebral birth injuries, either obstetric or spontaneous, may produce a similar picture for the first few days, and be characterized only by lassitude and bright green vomitus. They are readily differentiated from atresia but are more difficult to separate from malrotation with volvulus. As a rule cases of cerebral injury show but little distention of the stomach and duodenum on the plain roentgenogram, while gas in the intestine and colon, although scanty, tends to be normally distributed.

The remainder of the jejunum and upper ileum are obstructed only by atresia. Vomiting, grayish mucous stools, and x-ray evidence of distended loops down to the obstructed point lead to the diagnosis.

In the low ileum the problem may be difficult. Atresia and meconium ileus produce quite similar clinical pictures. Vomiting, distention and absence of meconium are common to each. Barium enema shows a tiny left colon in both instances. On palpation, however, a doughy feel in the lower abdomen may be elicited in

meconium ileus, and the packed loops may even be palpated. The x-ray film shows many distended coils in both; but in meconium ileus the meconium itself may occasionally appear as a finely granular shadow and small scattered air shadows, much smaller than the distended loops, may be seen. These are fairly diagnostic if found, and probably represent air bubbles which have been forced downward into the sticky mass without completely filling the lumen.

Atresia of the colon is extremely rare but gives a dramatic x-ray picture when it occurs. The colon distends immensely with air, sometimes filling half of the abdomen, and may be confused with pneumoperitoneum. This picture, combined with total absence of meconium, makes the diagnosis clearly apparent.

The left colon is the location for an interesting diagnostic problem which is overlooked more often than it should be, namely, the neurogenic obstruction of early Hirschsprung's disease. It is due to myenteric plexus deficiency, and microscopically it is evidenced by absence of ganglionic cells between the muscle layers. Its characteristic symptoms were outlined previously but it may again be stated that, provided the rectum is patent and in continuity with the gut above, there is no other congenital obstruction in early infancy which shows an enlarged left colon to barium enema and roentgenogram.

SUMMARY AND CONCLUSIONS

In view of the foregoing statements it becomes apparent that certain physical findings are of such diagnostic inference that a correct impression may often be reached at the bedside. In summary they may be stated thusly.

The baby who cannot breathe with closed lips probably has a choanal atresia.

The baby with a wet mouth and frothy mucus, who cannot swallow without coughing or becoming cyanosed, probably has atresia of the esophagus.

The child born with a small scaphoid abdomen which remains flat to percussion after six hours can only have atresia of the esophagus without fistula.

The baby with attacks of dyspnea and an exceedingly high respiratory rate may well have a diaphragmatic hernia.

The infant who persistently vomits green material probably has an atresia just beyond the ampulla, or a mid-gut volvulus.

The infant born with a distended abdomen which is flat to percussion may within a few hours develop further distention, local tympany overlying the flatness and a tense shiny skin. In such cases a diagnosis of prenatal obstruction with perforation, and a superimposed pneumoperitoneum, will seldom be wrong.

The infant with slow progressive abdominal distention, followed by vomiting and inadequate stools, and on whom a colon roentgenogram shows an enlargement of the left colon, has early Hirschsprung's disease.

The baby with imperforate anus has a ready made diagnosis for those who care to look. In males, if the imperforation is well above the skin level, a fistulous communication with the urinary tract, usually the posterior urethra, will co-exist more than 70 per cent of the time. Meconium in the urine makes this diagnosis certain; in any event it will be found and corrected during the abdominoperineal procedure. From the perineal approach it is often missed.

The size of the rectal sphincter is of importance, provided it has not been distorted by the first rude examining finger. It is always small and undeveloped in cases in which a congenital obstruction exists above it.

Vomiting is the most important symptom of all obstructions below the diaphragm; but its greatest value in differentiation depends upon whether it is clear, green or brown.

X-ray evidence, while our most valuable and conclusive diagnostic aid, can often be anticipated and at times clarified by bedside observation and deduction.

The application of these principles has been of proved value at the Childrens Hospital of Los Angeles, where the records for the past eighteen years show that 454 patients with alimentary tract obstruction have been operated upon, with an over-all survival rate of 53 per cent.

In conclusion, this wish for the unborn babies of the future is expressed; that some day, in every well equipped hospital, a plain roentgenogram of each baby will be taken at the end of twenty-four hours, and become a routine part of the chart. A portable x-ray unit connected with the nursery could make this possible with very little inconvenience. Obstetric fractures, skeletal abnormalities, atelectasis, transposition of viscera, diaphragmatic hernia, intestinal and esophageal atresias are just some of the abnormalities which could be diagnosed days in advance of the symptoms.

Today no patient is surrounded with more exquisite care than is the newborn, and to give him this extra protection would seem only his just due. Up to now, however, few if any hospitals have done so.

DISCUSSION

ORVILLE F. GRIMES (San Francisco, Calif.): It is certainly true that of all the surgical emergencies occurring in the newborn, congenital malformations of the gastrointestinal tract are by far the most common. Of these, the tracheo-esophageal fistula, the imperforate anus and the meconium ileus are the main offenders. When such alimentary tract malformations occur, about 25 per cent of these individuals will have serious abnormalities elsewhere, such as congenital heart disease.

If the defect is an imperforate anus, the percentage of additional malformations will be even higher since there may very well be associated genitourinary abnormalities. Before the definitive repair of an imperforate anus is attempted, an evaluation of the genitourinary system might well be made. Whenever reasonable doubt exists regarding the advisability of an immediate definitive repair of an imperforate anus, a simple decompressing procedure should be accomplished.

The differential diagnosis of alimentary tract obstruction in the newborn is of great importance since early diagnosis and prompt surgical intervention may prevent rupture of the proximal bowel which may occur early in the course of the obstruction. The distention proximal to the obstruction leads to ischemic necrosis of the bowel wall and subsequent perforation. Peritonitis in this situation is invariably fatal.

The practical factors which Dr. Nichols mentions are of great value in the diagnosis of these conditions. However, although the necessity for surgery is evident by the evaluation of physical and x-ray findings, an accurate diagnosis often cannot be established until surgery is accomplished.

B. P. MULLEN (Seattle, Washington): I would like to emphasize the anomalies of malrotation of the mid-gut loop producing obstruction. A brief discussion of the embryology will help bring to mind the development of these anomalies. As the mid-gut loop grows it is extruded through the umbilical ring into the umbilical cord due to the fact that there is not enough space in the cavity to contain it as the liver is very large at this time. It continues to grow as a physiologic umbilical hernia until the abdominal cavity has enlarged to the point where it can contain all of the loop. The small intestine during this stage elongates and the cecum becomes thickened and the enlarged cecum hangs up in the sac until the last. The small intestine is reduced, first producing normal rotation of the mid-gut loop which is in a counter-clockwise direction. However, when the umbilical ring is quite large, sometimes the entire mid-gut loop reduces simultaneously and instead of a counter-clockwise rotation a clockwise rotation results. Two types of obstruction may occur as a result. First, the entire mid-gut loop suspended from the narrow duodenal colic isthmus may become completely rotated one or more times producing a volvulus. None of the normal fixations of the intestine occur in this condition. Strangulation may occur or it may not actually strangulate, and the patient may survive with this condition until adult life. The other anomaly which I am most interested in is known as retroposition of the transverse colon. In this condition there is also a clockwise rotation but only of ninety degrees. The cecum and the transverse colon rotate behind the duodenum. The cecum may become wholly or partly attached in its normal position but the transverse colon becomes located behind the duodenum. As the root of the small intestine becomes adherent to the posterior abdominal wall in an oblique manner from the origin of the superior mesentery vessels to the cecum, it forms a tunnel about the transverse colon. The remainder of the large bowel attachments are normal. The obstruction is produced by a narrowing of this canal or tunnel, or by a volvulus of the cecum. I have two such cases I intend to report which were found incidentally at the time of a gastric resection. This condition is extremely rare. To date I have found only fourteen cases reported in the literature and these two cases will be the fifteenth and sixteenth. To the differential diagnosis of the closed loop obstruction as a result of a competent iliocecal valve plus complete obstruction distally due to a tumor or volvulus must be added the anomaly of retroposition of the transverse colon. Neither of my two cases had obstruction.

I believe that a resection of the right colon with anastomosis of the ileum to the transverse colon end-to-end beyond the tunnel and removing the cecum and extracting the part within the tunnel would be the procedure of choice. However, in cases of volvulus in which the attachment has not occurred, the procedure would be to simply unwind the volvulus with or without attaching the cecum in its normal position. This has been done successfully.

J. NORTON NICHOLS (closing): Dr. Grimes is, of course, correct in his statement that many of the abdominal conditions cannot be accurately differentiated until laparotomy is performed. A goodly number will still remain in which the true state of affairs can be suspected, and the observer alerted to the possibilities and dangers. This in itself makes careful observation worth while, for there is no preventable factor which increases mortality more than a delayed diagnosis.

I wish to express my thanks to those who have taken part in the discussion.

Experience with Ligation of the Pancreatic Ducts in the Treatment of Chronic Relapsing Pancreatitis

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IT is the purpose of this paper to present experience with six patients suffering from chronic relapsing pancreatitis who were treated by ligation of the pancreatic duct or ducts.

Experience at Wadsworth Hospital in the past has indicated that for the most part none of the surgical procedures advocated for the relief of symptoms of chronic relapsing pancreatitis in the absence of biliary tract disease, short of actual excision of the pancreas, has been invariably effective. Procedures most commonly advocated alone or in combination include: vagotomy, sympathectomy, sphincterotomy, gastroenterostomy with or without gastric resection, transplantation of the common bile duct, and caudal pancreatico-dochojejunostomy. The fact that so many procedures are advocated for the relief of this disease would seem to indicate that none is particularly effective. All of the surgical measures so far devised, short of excision, have had as their aims relief of assumed major duct obstruction, suppression of exocrine activity, or interruption of nerve fibers bearing pain impulses.

Neither the actual cause nor the pathologic physiology of chronic relapsing pancreatitis is well understood. It is probably safe to say, however, that breakdown of the ductal system with the consequent release of activated enzymes into the pancreatic stroma is essential to the production of the disease. In chronic relapsing pancreatitis it is the presence of episodes of severe pain which make the disease subject to surgical intervention. It would appear that the pain is the result of stromal digestion and irritation by activated enzymes which have escaped from the ductal system.

Rich and Duff[1] have reported the most extensive pathologic study of chronic relapsing pancreatitis and indicate that the only consistent finding of etiologic significance is the presence of epithelial metaplasia resulting in the obstruction of small ducts draining lobular groups or sections of the gland. Surgical measures available today cannot hope to relieve such obstructions in the duct system at this level. The common channel theory of the etiology of chronic pancreatitis has not been satisfactorily substantiated. When subjected to the therapeutic test of sphincterotomy in our hands and in the experience of others, with the exception of the original advocates of the method,[2] it has been repeatedly ineffective. The surgeon called upon to treat the patient suffering pain due to chronic relapsing pancreatitis consequently has the choice of trying numerous procedures about which many claims are made (but which may not stand the test of time) or of settling the issue squarely, once the indication is great enough, by excising the gland or applying some measure designed to destroy permanently the exocrine activity of the pancreas.

The case histories presented herein represent patients suffering from chronic relapsing pancreatitis who have had multiple surgical procedures as well as extensive medical management applied to their problem. None had obtained significant or lasting relief of his pain. Pancreatic duct ligation was applied as a possible alternative before subjecting the patient to actual glandular excision, in the hope of preserving the endocrine function of the pancreas but at the same time destroying the exocrine function responsible for the disease.

There is considerable experimental evidence to indicate that ligation of the pancreatic duct in normal animals routinely does destroy the exocrine function of the pancreas.[3–6] It is attended by almost no mortality, does not

produce acute pancreatitis and does not necessarily produce diabetes. In fact the discovery and isolation of insulin was based upon this premise, which had been tested and proved before as well as since the monumental work of Banting and Best in 1922.[7] Since that time various experimental animals with the exocrine portion of the pancreas destroyed by pancreatic duct ligation have been used extensively in research.

Experimental pancreatitis for the most part can be produced only by exerting enough retrograde pressure on the duct system to produce its rupture. Once rupture occurs, the enzyme secretions are apparently activated, escape into the surrounding stroma, digest and irritate the intrinsic tissue, and produce a chemical inflammation which may progress through the stages of hyperemia, edema, suppuration, hemorrhage and necrosis. Regression with healing, repair and return of function may occur if the initial process does not progress too far.

There appear to be two main causative factors for human pancreatitis, i.e., duct obstruction and episodes of exocrine hypersecretion producing sufficient pressure behind the obstruction to rupture the ductal system. The exocrine cells of the normal unstimulated pancreas of the experimental animal do not produce enough secretory pressure to rupture a totally obstructed duct system. Pancreatitis consequently does not develop because the exocrine cells atrophy when their normal function is prevented.

In the following six cases acute pancreatitis did not follow ligation of the pancreatic ducts and there is some evidence to indicate that pancreatic exocrine secretion was either depressed or destroyed in those cases in which terminal ligation of the entire ductal system was thought to be complete. Experience with the six cases is presented as a preliminary report in the belief that the concept may have therapeutic merit and in the hope that others will find the results sufficiently challenging to test the actual value of the procedure.

CASE REPORTS

CASE 1. B. L., a twenty year old housewife, entered Harbor General Hospital (No. 648057) on April 8, 1952, complaining of severe epigastric and back pain, along with intermittent vomiting of bright red blood and the passage of tarry stools for the preceding five hours.

During the previous two years she had gained excessive weight, starting at 130 pounds and going up to 207 pounds. Fourteen months before admission she complained of attacks of pain in the right upper abdominal quadrant, and vomiting. For several months she had been treated by osteopaths and on November 15, 1951, cholecystectomy had been performed. Because of severe epigastric and back pain complicated by attacks of massive hematemesis and hematochezia, the patient had been subjected to four additional laparotomies at osteopathic hospitals during the next five months.

On admission to Harbor General Hospital she again complained of unbearable epigastric and back pain, vomited bright red blood, and was in severe shock. Her serum amylase was 530 units, the normal for the laboratory being 320 units or less. Conservative management failed and at her sixth laparotomy the entire bowel was found to be filled with blood. The pancreas showed both acute and chronic inflammatory changes. No bleeding source was discovered and subtotal gastrectomy was performed. Bleeding started again during completion of the gastroenterostomy, and on further exploration was found to consist of a jet of blood spurting from the duct of Wirsung. Bleeding was halted by ligating the superior pancreaticoduodenal artery. The patient had a very stormy postoperative course and was discharged home only to be re-admitted six weeks later again vomiting blood, again in shock, and complaining of severe epigastric pain. Serum amylase at this time was not elevated. Immediate laparotomy for the seventh time was required to save her life. The source of bleeding was again the duct of Wirsung. Subtotal pancreatectomy was not feasible because of extensive inflammatory changes. The duct of Wirsung was consequently isolated, ligated and interrupted by dissecting the duodenal stump off the head of the pancreas until the duct of Wirsung was reached. (Fig. 1.) In the course of this procedure it was thought very likely that the duct of Santorini was ligated although it was not definitely identified. The duct of Wirsung was dissected free and found to enter the ampulla through a common channel with the common duct. The duct of Wirsung was interrupted and doubly ligated.

A portion of the duodenal stump was resected because of poor viability and a duodenal stump closure was finally accomplished along with closure of the duodenotomy. A T-tube was reinserted in the common duct and the wound was closed. The patient withstood the procedure well and was returned from the operating room in fair condition.

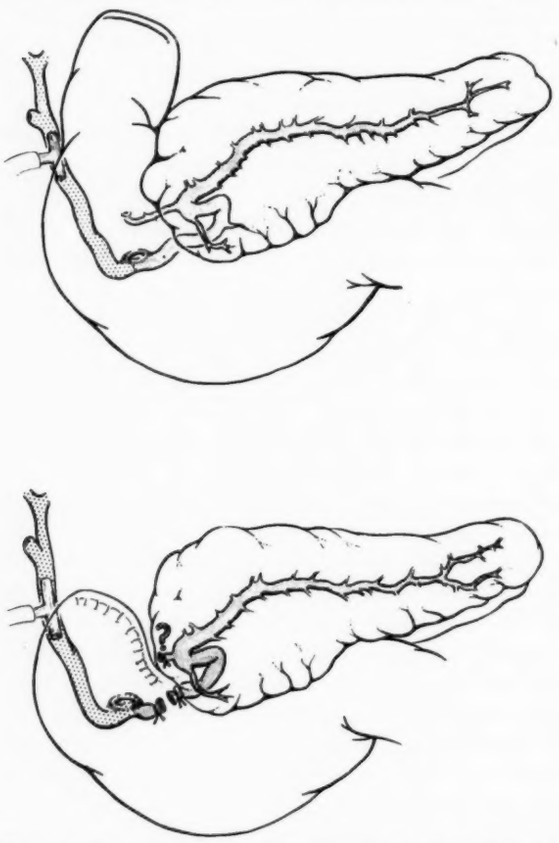


FIG. 1. Case 1. Method of pancreatic duct ligation. Performed by extraduodenal method. Duct of Santorini probably ligated. Common channel present.

Postoperatively, the patient's course was surprisingly benign. She complained of occasional back pain in the first few days immediately following the operation. She was maintained on large doses of antibiotics, atropine and pancreatin orally, the latter drug was given in the hope of suppressing the stimulus of the pancreas. On the second postoperative day serum amylase was 450, but in the next several days it dropped far below the normal of 320 units. She experienced a moderate amount of back pain and deep epigastric pain during the first three days after duct ligation, but this pain gradually subsided. In the subsequent few weeks the patient noted occasional twinges of back pain. Ten weeks after ligation she was readmitted complaining of moderate epigastric pain radiating into the back. There was no evidence of bleeding, but slight nausea and one episode of vomiting occurred. Serum amylase at that time was 523 units. She was hospitalized for two days and treated with pancreatin and banthine, after which she obtained complete relief. She is now thirty-one months postoperative, weighs 136 pounds and is asymptomatic.

The patient takes 20 gr. of pancreatin with meals for enzyme replacement at somewhat irregular intervals. Unfortunately, she could not be prevailed upon to come to the hospital for follow-up laboratory studies.

Comment: It is believed that this patient suffered from chronic relapsing pancreatitis complicated by erosion of an intrapancreatic artery which hemorrhaged into the duct system. Complications of pain and bleeding were resistant to cholecystectomy, common duct exploration, subtotal gastric resection and arterial ligation. In this case, ligation of the pancreatic ducts was a heroic measure dictated by circumstances but with apparent cure not only of the recurring hemorrhage but also of the pain from pancreatitis as well. Endocrine pancreatic function has been preserved. Duct ligation has not resulted in debility to the patient even though replacement therapy has been sporadic.

CASE II. J. R. B., a thirty-two year old man, was first admitted to Wadsworth General Hospital (No. 21274) on December 16, 1950, because of severe abdominal pain, nausea, vomiting and shock. His serum amylase was found to be elevated and a diagnosis of acute pancreatitis was made.

Past history revealed severe chronic alcoholism and a serious, long-standing depressive psychosis, which apparently started during the war when the patient went through several combat episodes while in the navy.

The patient's initial episode of pancreatitis responded to conservative therapy but in the course of the next two months he suffered repeated severe attacks of abdominal pain with only one- to two-day asymptomatic intervals. On February 16, 1951, at laparotomy the pancreas showed induration and nodularity consistent with the clinical diagnosis of chronic relapsing pancreatitis. A caudal pancreaticodocho-Roux-en-Y jejunostomy was performed. Aberrant pancreatic tissue in the mesentery of

the upper jejunum was resected. Both the pancreas and aberrant tissue showed chronic pancreatitis.

The patient was somewhat improved for three months, but then began having continual abdominal pain again. On June 26th cholecystectomy and sphincterotomy were performed. The pancreatico-docho-jejunostomy was found to be patent. A common channel at the ampulla of Vater was present and a patent duct of Santorini was demonstrated.

After two months the patient, who had continued his drinking, again began having chronic abdominal pain. Seven months following his second operation, bilateral splanchnicectomies were performed in two stages. Pain then became localized to an area between the scapulas in the back. Serum amylase determinations showed periodic elevations and he required increasing narcotics for his pain.

He was offered and accepted elective pancreatic duct ligation. On October 29, 1952, a third laparotomy was performed. The previous Roux-en-Y jejunostomy to the tail of the pancreas was inspected. The pancreatic duct was thought to be entirely stenosed and healed over; consequently, the anastomosis was taken down, the segment of Roux-en-Y jejunum was resected and bowel continuity re-established with an end-to-end enteroenterostomy. The duct of Wirsung, located through a duodenotomy, was found to open separately into the duodenum at a distance of about 3 to 4 mm. from the opening of the common duct, indicating that the previously performed sphincterotomy had been entirely successful in destroying any common channel between the bile and pancreatic ducts, since before sphincterotomy a common channel for the common duct and pancreatic duct was described. The duct of Wirsung was ligated transduodenally by making a second duodenotomy through the posterior wall of the duodenum paralleling the pancreatic duct. The duct of Wirsung was doubly ligated and interrupted. (Fig. 2.) No attempt was made to search for the duct of Santorini since it was thought that the patient might benefit from ligation of the main duct alone. A T-tube was placed in the common duct, the duodenotomy closed and the patient returned to his room in fairly good condition. The patient's serum amylase was 315 on the second postoperative day, 114 on the third and 110 on the fourth. The fifth postoperative day it was 98 and remained thereafter at a level between 40 and 60 units. The patient had some pain in the abdomen but thought soon after operation that he was having less pain in his back. He had two bowel movements on the fourth postoperative day and was placed on surgical fluids. In the course of the next six days, pain gradually developed in the left upper abdominal quadrant. He stated that the pain was entirely different from previous pancreatic disturbance. He began running a spiking fever and had trouble with increased vomiting. Roentgenograms showed an elevation of the left diaphragm and examination gave an impression of fullness to palpation and flatness to percussion in the left upper quadrant. A diagnosis of subphrenic abscess was made. Incision and drainage yielded pus with an amylase content of 500 units. The patient improved briefly but then a second abscess localized in the right lower quadrant, which was also drained thirty days after pancreatic duct ligation. Two days later he complained of tenderness in the left foot and leg. He received 200 mg. of depoheparin but died suddenly a few hours later of what clinically appeared to be a massive pulmonary embolism.

The gross description of the pancreas at postmortem examination was as follows: "The pancreas is of normal size and shape; the cut section reveals a hyperemic surface with the usual lobular pattern. It is somewhat indurated and nodular but there is no other evidence of pancreatitis visible. The portion of the pancreatic duct emptying into the ampulla of Vater shows dilatation to a circumference of 1.5 cm. and the duct terminates in a blind end, the result of a silk ligature. The dilatation extends into the substance of the pancreas for a distance of 3 cm., at which point the duct is lost. Examination of the tail of the pancreas reveals scarring and adhesions but no patent duct can be found."

The microscopic examination of the pancreas was as follows: "There is marked perilobular and intralobular fibrosis and many large areas have undergone postmortem autolysis. The islets of Langerhans are infrequent but many appear normal. The cystic structure is lined by flattened epithelium and fibrous tissue which is infiltrated by mononuclear cells. The picture is that of chronic fibrosing pancreatitis."

Examination of the abdominal cavity revealed extensive adhesions but no evidence of

pus. Apparently the two abscesses had been drained adequately. Both the entire right heart and the major pulmonary arteries were filled with large antemortem blood clots which appeared to have arisen from the vena cava and the veins draining the left lower extremity, undoubtedly representing the cause of death.

Comment: We believe it is fair to point out that subsequent to pancreatic duct ligation, this patient volunteered the information that he no longer had the pain which had caused him so much trouble before duct ligation. He began to eat and seemed to be improving, before the series of complications developed which finally took his life. Because the serum amylase content of the abscess fluid from the left subphrenic region was high, it was postulated that pancreatic duct ligation had resulted in reopening of the duct at its exit from the tail of the pancreas. The sterile pancreatic enzymatic abscess which followed had probably been preceded by enzymatic peritonitis. The abscess in the left subphrenic region was drained successfully with relief. The process localized secondarily in the pelvis in an abscess which was also drained, following which the patient began passing gas, started to eat and noted that most of his pain was gone. At no time did pain of the preoperative nature recur to any extent that could be elicited. Admittedly no real conclusions can be drawn in this situation because of the patient's unfortunate demise from the venous thrombosis which was found and treated too late. In retrospect it was believed that the problem might well have been averted if at the time the caudal pancreatico-docho-jejunostomy was taken down a set of sutures had been placed in the tail of the pancreas to assure ligation of the duct at that end.

Certainly the patient gained no relief from caudal pancreatico-docho-jejunostomy (the patency of which was subsequently proved), cholecystectomy, sphincterotomy, common duct drainage or bilateral splanchnicectomy.

Significantly, the patency of the caudal pancreatico-docho-jejunostomy still existed four months after it had been performed. The sphincterotomy was effective in destroying the common channel between the common bile duct and pancreatic duct as well as the sphincter. No obstruction of the duct was found at the time of duct ligation.

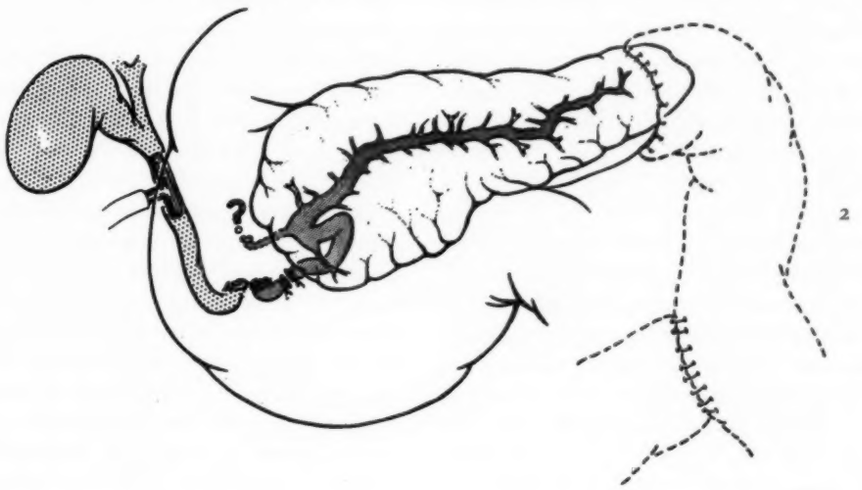
The fact that biopsy of a fragment of aberrant pancreas from the jejunal mesentery at the time of the patient's first laparotomy showed chronic pancreatitis is a phenomenon deserving sober reflection and explanation.

Case III. E. E. H., a thirty-two year old white machinist, was admitted for the sixth time to Wadsworth Hospital (No. 29990) on October 6, 1952. From the time of his initial episode in 1942 the patient had had twenty to thirty bouts of acute pancreatitis. His present attack began forty-eight hours prior to admission with the sudden onset of severe epigastric pain radiating to the back, with nausea and vomiting. This illness was preceded by no particular dietary indiscretion or alcoholic intake. Prior attacks occurring every six to twelve weeks for the previous two to three years had been of a similar nature but there was never a period of more than three to four months between attacks. A diagnosis of chronic pancreatitis was initially made in 1943 by exploratory laparotomy for an acute condition of the abdomen. In 1949 vagotomy, performed because of the intense pain, provided relief for a period of two years, but also resulted in the development of frequent abdominal distention and diarrhea with the passage of fatty, foul smelling stools. Because of a diagnosis of gastric retention subsequent to vagotomy, gastrojejunostomy was performed in 1951. Following this operation, diarrhea and abdominal distention ceased but pain recurred so that the patient believed that his chronic picture had reverted to prevagotomy status.

Physical examination at the time of admission showed a well developed somewhat undernourished white man who appeared acutely ill. His weight was 122 pounds. The abdomen showed localized epigastric tenderness and rigidity in the area of his three old operative scars, but no masses or organs were palpated. Stools were tan and showed moderate increase in fat content and in digested food but no occult blood. Serum amylase was 84 units, the normal for this laboratory being 80–120 units. Abdominal films showed numerous calcifications in the pancreatic area. An upper gastrointestinal series showed a normally functioning postoperative gastroenterostomy with no evidence of marginal ulcer.

When the patient failed to respond to conservative medical therapy in the course of the next six weeks, he was offered pancreatic duct ligation which he accepted. On November 20,

1952, transduodenal interruption and ligation of the duct of Wirsung were performed. (Fig. 3.) No search was made for the accessory pancreatic duct. On palpation the pancreas showed induration and nodularity but no obvious acute inflammation. The duct of Wirsung was admittedly both openings were surrounded by the sphincter of Oddi. The duct of Wirsung was consequently ligated and interrupted through an incision in the posterior wall of the duodenum, and a T-tube was left in the common duct. Serum amylase determinations



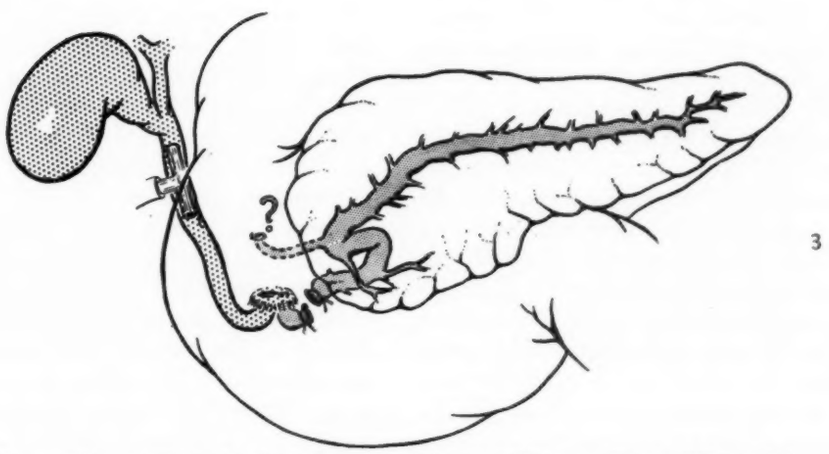


FIG. 2. Case II. Transduodenal ligation of duct of Wirsung. Duct of Santorini shown to be patent at previous operation but not visualized at time of duct ligation. Common channel destroyed by previous sphincterotomy.

FIG. 3. Case III. Transduodenal ligation of duct of Wirsung. Duct of Santorini probably patent. Pancreatic duct and common bile duct surrounded by sphincter of Oddi, but separated by septum.

located only after a common duct exploration was carried out. The duct of Wirsung entered the duodenum through the ampulla of Vater. There was a definite bridge of tissue between the common duct and the pancreatic duct, extending all of the way to the lumen of the duodenum, so that in actuality there was a separate opening of the pancreatic duct and common duct into the duodenum, although made daily after surgery were as follows: November 21st, 47 units; November 22nd, 32 units; November 23rd, 24 units; November 24th, 60 units; November 25th, 48 units; November 26th, 24 units; November 27th, 32 units. These values are far below normal, but represented no rise from the average level of serum amylase which the patient ran preoperatively. The patient was able to take a

bland diet by the fifth postoperative day. Fourteen days postoperatively he was allowed to go out on pass, but returned one week later complaining of severe epigastric pain radiating through to the back. No relief was obtained by unclamping the T-tube although a free flow of bile was obtained. The patient's serum amylase was 50. Cholangiogram revealed no evidence of obstruction to the common bile duct and the T-tube was removed on December 8, 1952. The patient appeared to be benefited by 20 gr. of pancreatin taken three times daily. He was discharged from the hospital and advised to continue taking the pancreatin as prescribed.

Since that time the patient's follow-up has been difficult. He has repeatedly entered various Veterans Administration Hospitals in other parts of the country, always with a diagnosis of chronic relapsing pancreatitis and always refusing any significant treatment other than medication, which usually was of his own choosing. He later returned to this area and was admitted to Wadsworth Hospital again on June 23, 1954, and discharged on July 21, 1954. Repeated psychiatric evaluations indicated that a significant portion of the patient's condition could be traced to mental abnormality.

Because the patient's pain problem remained unsolved, he was offered a total pancreatectomy. He was receptive to this suggestion, but insisted the operation be performed at the Thayer Veterans Administration Hospital. He was therefore discharged with the recommendation that he be transferred there, if possible. At present no further follow-up on him has been available.

Comment: It has been our experience that most of these patients represent a severe psychiatric problem. However, it has not been possible to state which has occurred first, the psychiatric problem or the chronic relapsing pancreatitis. The diagnosis of chronic relapsing pancreatitis was confirmed by the two laparotomies, abnormally low serum amylases and calcification in the region of the pancreas. It is our belief that this particular patient was left with a patent duct of Santorini which continued to drain his pancreas after ligation of the duct of Wirsung. This belief is supported by lack of elevation in serum amylases taken daily after the pancreatic duct ligation. In this case, the duct of Santorini may have assumed the function of draining the entire pancreas.

Case IV. R. A. B., a forty year old man was admitted to the Wadsworth Hospital (No. 17688) on September 25, 1952, with a fifteen-year history of chronic alcoholism. He had been treated on three previous occasions at the Brentwood Neuropsychiatric Hospital for alcoholic psychosis and delirium tremens. In July, 1950, following an episode of drinking, he was readmitted to the surgical service because of severe upper abdominal pain which radiated through to his back. Serum amylase at that time was 600 units, the normal for this laboratory being 80–120 units. Pancreatic calculi were visible roentgenographically. A cholecystogram was normal and a gastrointestinal series revealed only a small hiatal hernia. The patient was treated medically and improved. One month later he was readmitted because of upper abdominal pain, at which time section of the sphincter of Oddi was performed. At the time of surgery the gallbladder appeared normal. Following discharge he continued to have epigastric distress and two months later was again admitted with a serum amylase of 480 units. Gastric analysis showed a marked increase in free hydrochloric acid and cholecystogram revealed a non-functioning gallbladder. At the time of the sphincterotomy, the surgeon noted that the common duct and the duct of Wirsung entered into a common channel in the ampulla of Vater. The patient symptomatically improved on banthīne, but during the first eight months in 1951, he was hospitalized four times because of exacerbations of upper abdominal pains which usually radiated through to his back or to his left shoulder.

In September, 1951, vagotomy and antral resection of the stomach with a Polya-anastomosis were performed in the hopes of relieving the pain from chronic pancreatitis. Following operation the patient continued to have frequent bouts of right upper quadrant and epigastric pain with nausea and vomiting. He was readmitted in February, 1952, with acute pancreatitis and again treated medically. On September 25th he was readmitted because of recurrence of pancreatitis. Following admission a gastrointestinal series, glucose tolerance test and prostigmine test were all normal. Serum amylase was 210 units. He was treated for twenty-four hours with gastric suction, with relief of symptoms. The patient was presented to the surgical conference where it was recom-

mended that he be considered for ligation of the pancreatic ducts. On December 4th the duct of Wirsung was visualized by duodenotomy in the right anterolateral surface of the duodenum. The ampulla of Vater could not be positively identified because of the rather marked degree of nodular enlargement of the head of the pancreas which protruded into the duodenum. The common duct was consequently explored and the ampulla of Vater identified. The ampulla appeared to be atrophic so that there was no actual papillary projection in the duodenum. On careful inspection pancreatic juice was seen exuding from a 2 mm. pancreatic duct orifice in the duodenum located at least 6 mm. medial to the opening of the common duct into the duodenum. A posterior duodenotomy was performed over a probe inserted in the duct of Wirsung which was ligated and divided. (Fig. 4.) The duct of Santorini could not be identified. Serum amylase levels in the immediate postoperative period were as follows: December 5th, 66 units; December 6th, 74 units; December 7th, 70 units; December 8th, 90 units; December 9th, 79 units; December 10th, 66 units; December 11th, 48 units; and December 13th, 109 units.

He was placed on a soft diet by the seventh postoperative day, and was also given banthīne and pancreatin. He continued to do well until December 29th when he went out on pass for two days and had a small amount of alcoholic intake, which he stated he took to test his operation. The result was recurrence of pancreatic pain. Following subsidence of that episode he continued to do fairly well, although an analysis of the duodenal aspirations revealed the presence of pancreatic juice, which may indicate that the duct of Santorini is patent.

After discharge his alcoholic habits returned and shortly he was readmitted to the psychiatric hospital where he spent several months under treatment and observation. Six months after duct ligation, he was readmitted to the hospital in a cyanotic and comatose condition. Blood pressure was not obtainable. Significant laboratory findings included a serum amylase of 288. Following conservative supportive therapy the patient improved and, when questioned later, admitted that he had been steadily drinking in his room during the previous week and had not eaten for two days preceding his admission. In the course of treatment sugar was noted in the urine, and it was necessary to place the patient on 20 units of NPH insulin, which was later reduced to 15 units. He was sufficiently improved to be discharged from the hospital on August 10, 1953, at which time he was taking NPH insulin 15 units daily, pancreatin gr. 60 daily, and atropine gr. $\frac{1}{100}$ four times a day. He was readmitted on December 18th because of an inguinal hernia, which was operated upon and from which he recovered without incident. Since that time he has had no further admissions and reports that he still has intermittent episodes of pain usually brought on by use of alcohol.

Comment: Postoperatively this patient did not show a rise in serum amylase level, and since subsequent analysis of duodenal aspirations demonstrated evidence of pancreatic secretions, it is highly probable that this patient has a patent duct of Santorini which has taken over drainage of his pancreas.

Case v. A. B. W., a fifty-nine year old retired telephone lineman, was admitted to Wadsworth Hospital (No. 20566) on February 11, 1954. He gave a five-year history of serious gastrointestinal complaints and had been admitted to this hospital at least five or six times for these symptoms since 1951. His symptoms consisted primarily of intermittent episodes of epigastric and left upper abdominal quadrant pain associated with nausea and vomiting. Radiation of the pain to the back and hematemesis were noted on several occasions. He had had repeated gastrointestinal series, only one of which was considered suggestive of duodenal ulcer; in the others a normal duodenal bulb was found although there was evidence of duodenal irritability. A presumptive diagnosis of duodenal ulcer and anxiety neurosis was made. The patient after treatment with antacids, diet and antispasmodics noted little more than transient relief of symptoms. His present hospital admission was preceded by a similar episode of epigastric pain of five days' duration. None of the numerous serum amylase determinations previously obtained was above 70 units, normal for this laboratory being 80–120 units. He admitted a regular alcoholic intake of rather large amounts over the course of many years. There was evidence of emotional instability and an inadequate personality.

Physical examination showed a temperature of 98.6°F., blood pressure 95/80, pulse 108. The

patient was a thin apprehensive man who appeared dehydrated and complained of severe pain. His abdomen was distended and somewhat tympanitic. Peristalsis was normal and there was tenderness in the epigastrium and right upper quadrant. Laboratory data at that time was not remarkable except for a serum amylase of 422 units.

The patient began to run an elevation of temperature, became irrational and developed hallucinations. Progressive abdominal distention associated with pain and tenderness in the epigastrium and right upper quadrant appeared. A diagnosis of either acute pancreatitis or perforated duodenal ulcer was made. On February 18th exploratory laparotomy revealed the entire pancreas to be firmer than normal and enlarged throughout. It was excessively indurated and there were scattered areas with whitish yellow, firm ½ to 1 cm. nodules adjacent to the pancreas which were reported to be soap spots. Some scarring was noted between the duodenum and liver but there was no evidence of duodenal ulcer. The patient's postoperative course was somewhat stormy because of increased psychotic mani-

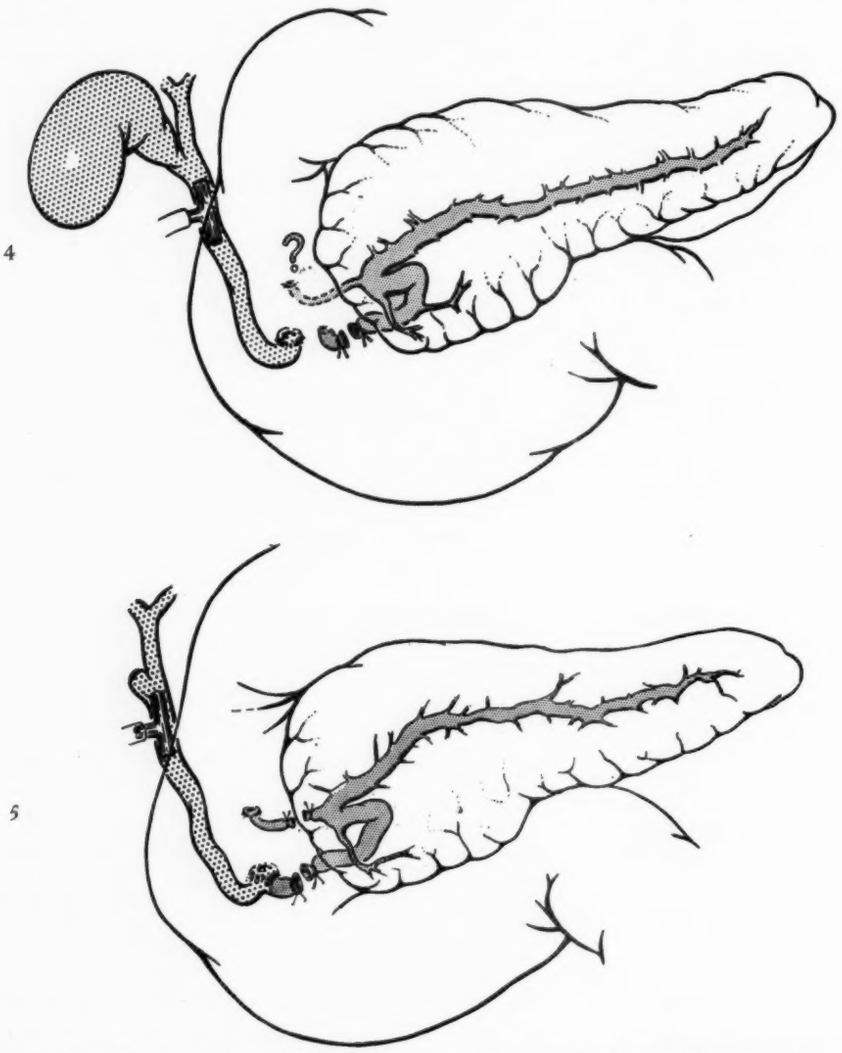


FIG. 4. Case IV. Transduodenal ligation of pancreatic duct of Wirsung. Duct of Santorini probably patent. Original common channel separated by previous sphincterotomy.

FIG. 5. Case V. Transduodenal ligation of both the duct of Wirsung and of Santorini. Common channel present.

festations, but his abdominal symptoms continued unabated. An upper gastrointestinal series performed on March 17, 1954, was normal.

On re-evaluation it was believed that the patient's symptoms, including those of the previous five years, were due to chronic pancreatitis. For this reason a pancreatic duct ligation was suggested which the patient accepted. At operation on April 7, 1954, a common channel between the common duct and the duct of Wirsung at the ampulla of Vater was noted. There was no obstruction at the ampulla of Vater. The duct of Wirsung was doubly ligated and divided transduodenally. (Fig. 5.) Exploration then revealed a patent duct of Santorini, approximately 1.5 cm. above the ampulla of Vater. This was also ligated by a duodenotomy through the posterior wall of the duodenum. The gallbladder was removed, the common duct thoroughly explored and a T-tube left in place. Postoperative serum amylase determinations were as follows: April 8th, 710 units; April 9th, 356 units; April 10th, 93 units; April 11th, 48 units; April 12th, 144 units; April 13th, 96 units; April 15th, 64 units; April 26th, 360 units; April 28th, 368 units; May 3rd, 144 units; May 9th, 157 units; May 24th, 90 units; June 14th, 76 units; July 2nd, 16 units. From April 10th through the 25th his pain steadily decreased. On April 26th he suffered an exacerbation of pain and showed an elevated amylase for some time thereafter. He then began to feel better and, although still complaining slightly, his amylase on July 2nd was only 16 units. He had been maintained since surgery on daily doses of pancreatin to replace the pancreatic secretions. Psychiatric consultation indicated that the patient had a strong functional component to his symptoms. In the course of the succeeding weeks, the patient's pain seemed to be improved although he still complained of pain in the area of the wound.

He was seen at varying intervals in the follow-up clinic, invariably complaining and intoxicated, but never with convincing evidence of abdominal tenderness, or even consistent localization of tenderness.

When last seen on January 17, 1955, eight and a half months after complete pancreatic duct ligation, it was immediately apparent that he had gained weight since his last operation. He stated that his weight had increased from 120 to 132 pounds. He said his only discomfort consisted of occasional twinges of pain in his "gallbladder" and pointed vaguely to his right lower abdominal quadrant. He stated that he had decided to give up alcohol, because he was out of jail on bond awaiting trial for a recent arrest for drunken and disorderly conduct.

Comment: This patient is believed to have had a complete ligation and interruption of the pancreatic duct system. In spite of ligation, several weeks were required for pancreatic function to be suppressed. It is clinically apparent that this patient has been benefited in spite of continued stimulation of his pancreas by excessive alcoholic intake. Unfortunately the main result of the operation may well be that the patient may now be able to drink himself into a state of delirium tremens or alcoholic cirrhosis without intervening episodes of pancreatitis.

Case VI. E. O., a divorced auto worker, age forty-one, was first admitted to Wadsworth Hospital (No. 49574) on October 8, 1954. He stated that approximately fifteen months before admission he began to have sharp and stabbing right upper abdominal quadrant pain radiating through to the back which would last for months at a time. He had been hospitalized for this pain seven times during the previous year with a diagnosis of pancreatitis. He had had nausea and vomiting on occasion, although he had never noted black stools, dark urine or jaundice. His average alcoholic intake consisted of one-half pint of whiskey and about a case of beer a week. The day before admission to Wadsworth he had been discharged from the Ventura County General Hospital with a clamped T-tube in the right upper quadrant.

A record obtained from the General Hospital in Ventura County stated that the patient had been admitted on August 12, 1954, complaining of episodes of abdominal pain and vomiting for the previous eighteen months. Two days prior to admission and following an alleged drinking bout steady periumbilical pain developed and the patient was unable to tolerate any food whatsoever. On admission to the hospital there were no special abnormalities except for the abdomen which showed upper abdominal prominence and generalized mild tenderness in the epigastrium. There was some guarding in this area, but no definite rigidity. Serum amylase was 237 units; the normal for

that laboratory being 60–150 units. The patient was treated conservatively and apparently responded. A cholecystogram was negative. He was readmitted there on September 10, 1954, with the same complaints, and with an elevated serum amylase. On September 15th,

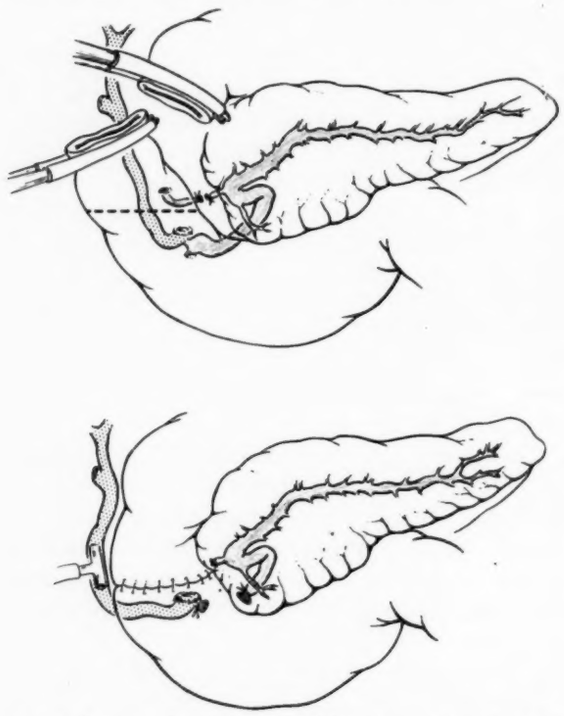


FIG. 6. Case VI. Extraduodenal ligation of ducts. Common channel present. Repair of duodenum by resection of devascularized portion and end-to-end anastomosis.

exploration revealed no abnormalities except that the pancreas was slightly firmer than usual. The gallbladder was not thickened and contained no stones, but was removed. The common duct was explored. Duodenotomy was performed and a section of the sphincter of Oddi was completed. Postoperatively the patient denied that he had obtained any relief from the operative procedure and was discharged home shortly before admission to Wadsworth.

On this admission, examination revealed a thin, well developed man in acute abdominal distress with a right upper quadrant scar and a T-tube in place. He showed well circumscribed, deep tenderness in the epigastrium. No organs or masses were felt and there was no spasm or rebound. The patient was subjected to a period of conservative treatment, observation and work-up for the next seven weeks. It was then suggested that ligation of the pancreatic ducts might be indicated since the patient had received no relief from his continual pain. On December 1st the duodenum was opened over the region of the ampulla of Vater, the common duct was explored, and a probe was passed into the duodenum. Extensive search did not reveal the location of the duct of Wirsung so that it was necessary to transect the duodenum completely about 2 cm. above the ampulla of Vater, at the level of the upper margin of the pancreas. It was then possible to dissect the duodenum downward off the head of the pancreas, until the duct of Wirsung was encountered. The duct was somewhat enlarged and indurated, but was found to have a free opening into the duodenum through two separate channels, one about 1 cm. inferior to the ampulla of Vater and the other opening directly into the ampulla. The latter had resisted discovery on direct exploration because of its angle and its position. The duct of Wirsung was consequently ligated and divided. (Fig. 6.) The non-viable portion of the duodenum was resected and end-to-end duodenoduodenostomy was performed. A T-tube was placed in the common duct and the abdomen closed. Preoperatively the patient's serum amylase determinations had not run above a high of 89 units, and averaged between 40–50 units. Postoperatively the patient's serum amylase determinations were as follows: December 2nd, 445; December 3rd, 254; December 6th, 77; December 10th, 34; with an increase to 118 on December 13th and a fall to 49 on January 9, 1955. The patient's postoperative course was one of continued pain until the fifth week when he noted that pain was decreasing, his appetite increasing, he required fewer narcotics, and wanted to go home.

Comment: This patient is considered to have had a complete interruption of the pancreatic duct system. He is too recently postoperative for any evaluation. It is hoped that his pancreatic function will finally be suppressed and his condition thereby improved.

OBSERVATIONS

In reviewing the literature, only one other case of elective ligation of the pancreatic ducts for chronic relapsing pancreatitis was found. This case was reported by Martin and Canseco in 1947.[8] The patient developed a pancreatic fistula postoperatively which drained for several days and then closed. Pain was relieved

and the patient appeared to have derived real benefit from the procedure for the follow-up period of ten months which was reported.

Of the six cases presented herein, the first patient has apparently been entirely relieved of her complaints through a follow-up period of thirty-one months. She had a complete ligation of the pancreatic duct system.

The second patient appeared to have obtained relief from his pancreatic pain for a three-week period, but his course was so complicated that no conclusions can be drawn. All forms of therapy applied to his problem failed.

The third patient has received no benefit to which he will admit. Since his duct ligation there has been no objective evidence of acute pancreatitis in the form of an elevated serum amylase. Since he showed no rise in serum amylase in the immediate postoperative period, it is possible that he has a patent duct of Santorini which drains his pancreas enough to allow its diseased exocrine function to continue. Admittedly his case might be considered as evidence that pancreatic function cannot always be destoyed by ligation of the duct of Wirsung alone or even that the pain of chronic relapsing pancreatitis is not a direct result of pancreatic exocrine function. The psychopathic over-lay in his case tends to confuse the issue.

The fourth patient, too, is far from a therapeutic triumph. His duct ligation may not have been complete since the duct of Santorini if present may be patent. He did not have a convincing rise in serum amylase in the immediate postoperative period. Nevertheless, for several months after duct ligation he gained weight and thought his pain was significantly less. However, he continued a high alcoholic intake, began to complain of further pain, was admitted once with an elevated serum amylase and has developed a mild case of diabetes which is an undeniable indication of progression of his disease.

The fifth patient is known to have had a complete ligation of the duct system of the pancreas since both the duct of Wirsung and Santorini were ligated and interrupted. Pancreatic exocrine activity, as indicated by episodes of amylase elevation and symptoms of pain, was present through the fourth postoperative week. Since that time the patient was seen at intervals in the follow-up clinic, invariably complaining and intoxicated, and never with convincing evidence of abdominal tenderness or amylase activity. His serum amylase level barely registers at 16 units. He has gained weight and he feels better than he did before operation. It is believed that the problem with his pancreas has been improved in spite of unrelenting alcoholic stimulation.

The sixth patient is still too recently postoperative to allow critical evaluation. At the time of this writing he had begun to eat, required less medication for pain, and his amylase level seemed to be dropping.

Thus, counting the case reported by Martin and Canseco, seven cases of pancreatic duct ligation for chronic relapsing pancreatitis are now on record; two have shown improvement, one has probably been improved, one received debatable benefit from ligation of the duct of Wirsung alone, one received no benefit from ligation of the duct of Wirsung alone, one is too recently postoperative to evaluate, and one death has occurred as a result of pulmonary embolism.

The six cases added to the literature in this report indicate that most of the other surgical procedures advocated in the past cannot always be expected to result in benefit. Sphincterotomy has been particularly unsatisfactory; bilateral splanchnic sympathectomy does not necessarily relieve the pain of this disease.

Numerous questions remain to be answered. Is the pain in chronic relapsing pancreatitis indeed due to exocrine activity in the gland? If pain is not due to exocrine activity, what is the explanation? If complete duct ligation will result in complete cessation of exocrine activity, how long an interval is required for activity to cease? Will duct ligation result in cyst formation?

On the basis of present experience it is believed that if duct ligation is to be used, every effort should be made to make certain that both ducts are ligated. Ligation of the pancreatic ducts in patients suffering from chronic relapsing pancreatitis has not precipitated acute pancreatitis. If the experience indicates nothing else, it can at least be said that duct obstruction alone is not the sole factor in the etiology of pancreatitis. By its very definition chronic relapsing pancreatitis is a disease characterized by remissions and exacerbations. Remissions in some instances may apparently last for months or years. Evaluation of any surgical procedure designed to relieve this condition will hence require a

long term follow-up program. It is possible that in the past too much attention has been directed to supposed beneficial results unsubstantiated by sufficient follow-up, and too little attention to subsequent remissions in cases subjected to operations of presumed value.

No claims are therefore made in this presentation. The longest follow-up of any of the cases presented is thirty-one months. Whether her relief is derived from remission or therapy remains a question.

It is however believed that the theoretic basis for pancreatic duct ligation in this problem is sound and that the results so far are sufficiently encouraging to warrant continued trial at least until a series large enough to permit statistical evaluation has been obtained.

This procedure is not recommended except as a possible alternative to pancreatic resection and then only if generalized pancreatic involvement is believed to be present. Trial of the method on an experimental basis is considered justified because of the mortality of pancreatectomy and the inevitable diabetes which will follow total pancreatectomy. Transduodenal pancreatic duct ligation technically is little more formidable than sphincterotomy once the orifices of the ducts are located. Extraduodenal pancreatic duct ligation is a more certain approach and the necessary partial duodenal resection and duodenoduodenostomy is little more difficult than gastroenterostomy.

It is believed that the actual solution to the problem may come from the chemistry laboratory in the form of an agent which would block the action of pancreatic enzymes or at least their intrapancreatic activation. In the meantime the pain of chronic relapsing pancreatitis will keep the disease in the domain of the surgeon who frequently seems to have little to offer the miserable patient but another operation of questionable theoretic value.

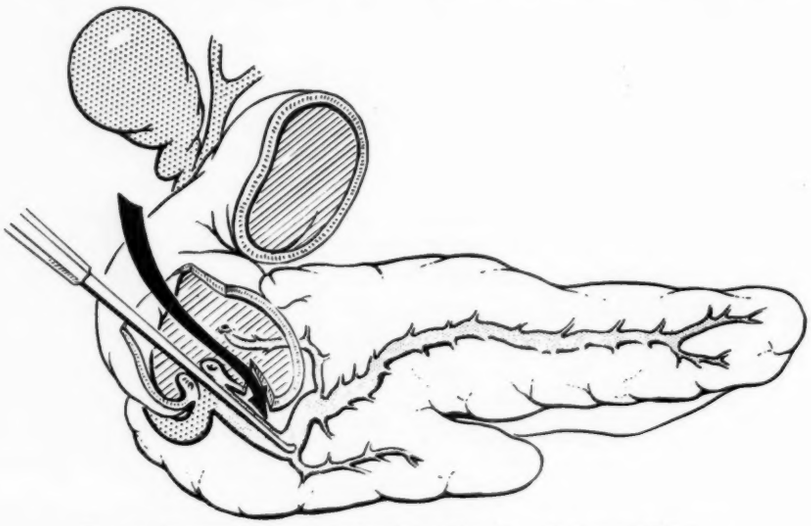


FIG. 7. Approach for transduodenal ligation of duct of Wirsung.

SUMMARY AND CONCLUSIONS

1. Experience with six cases of elective pancreatic duct ligation for chronic relapsing pancreatitis is presented.

2. One patient appears to have been relieved, one has probably been improved in spite of continued alcoholism, one received debatable benefit from ligation of the duct of Wirsung alone, one received no benefit from ligation of the duct of Wirsung alone, one is too recently postoperative to evaluate, and one death from pulmonary embolism occurred.

3. Duct ligation was performed transduodenally in four cases (Fig. 7) and extraduodenally in two cases.

4. The rationale of the procedure is based on the premise that pancreatic duct ligation will result in destruction of pancreatic exocrine activity and that the latter is responsible for the pain in chronic relapsing pancreatitis.

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DISCUSSION

John R. Paxton (Glendale, Calif.): I would like to preface my discussion with the fact that I have had no experience with the procedure presented.

Before discussing Dr. Cannon's paper, I think it might be helpful to review the clinical types of acute and chronic pancreatitis, so that we may have a more common understanding of the problem being discussed.

In type I the patient undergoes his first attack of acute pancreatitis; the acute process subsides and the patient remains well and asymptomatic.

In type II an acute attack develops; the acute phase subsides and the patient remains asymptomatic, but subsequently recurrent acute attacks develop. This recurrent acute pancreatitis type, in which the patient remains completely asymptomatic between attacks, is totally different in pathology and probable etiology than the type discussed by Dr. Cannon.

In type III usually the patient is a chronic alcoholic and repeatedly has episodes of acute alcoholism. He almost invariably has a marked psychopathic background, is a ne'er-do-well, refuses to accept responsibility, and has episodes bordering on maniac depressive psychosis. His history of psychiatric instability and alcoholism antedate for years his first onset of acute pancreatitis.

He also commonly will give a history of indigestion consisting of nausea, vomiting and mild epigastric pain, associated with bouts of alcoholism, commonly misdiagnosed as alcoholic gastritis for four or six attacks before his first major attack of acute pancreatitis develops. His first major attack is commonly severe, frequently presents evidence of extravasation of pancreatitic fluid in the epigastrium, and down either or both retroperitoneal gutters. Frank psychosis may develop on the third or fourth day of his illness, and usually the convalescent period is prolonged.

Following the acute episode he commonly has a persistence of mild epigastric symptoms in contrast to types I and II, then continues to have repeated acute attacks, some severe and some moderate, and a progressive increase in persistent symptoms between attacks; ultimately he may become a narcotic addict.

Pathologically types I and II show the characteristic evidence of acute pancreatitis, either with surgical biopsy, or at postmortem, but with little evidence of residual fibrosis, calcification, ductal hyperplasia, or multiple areas of cystic degeneration. All of which are commonly seen in type III.

Although little is actually known about the etiology of either acute or chronic pancreatitis, I think it is gradually becoming accepted by some students of the disease that there is a trigger mechanism outside the pancreas that initiates the acute phase of the disease. One is unprepared to state at this time whether this trigger mechanism be by excess cerebral cortical stimulation mediated through the anterior hypothalamus and in turn through the vagus nerve, with all the changes in muscle tonus, vascular supply, hypersecretion of stomach, duodenal and pancreatic secretions; or whether through the posterior hypothalamus and then through the adrenal cortex, with all of the above changes in stomach, duodenum and pancreas (as demonstrated experimentally by Porter, Movius and French); or whether the common channel theory of Opie, with or without stone or spasm of the ampulla, or papilla of Vater.

However with the above mentioned known pathologic changes in types I and II and if one accepts any or all of the foregoing concepts of etiology, it immediately becomes apparent that surgical interruption of these trigger mechanisms, whether by cholecystectomy, exploration of the common duct and transduodenal sphincterotomy, subtotal gastric resection, vagotomy with pyloroplasty and sphincterotomy, might explain the reported good results in these procedures in types I and II of the disease.

By the same reasoning one would not expect any of the foregoing procedures to be of any permanent value in type III of the disease due to the marked difference in pathology with ductal obstruction, metaplasia or hyperplasia, calcification with marked fibrosis, and even multiple areas of cystic degeneration.

With these pathologic changes and a continued exocrine function of the gland remaining, it seems illogical to expect surgical procedures such as

splanchnicectomy or sympathectomy to do other than possibly control the chronic type of pain of the disease, and not change the basic pathology.

Caudal pancreatico-docho-jejunostomy attempting to decrease pancreatic ductal pressure in a retrograde manner seems illogical with multiple areas of ductal obstruction throughout the entire pancreas. This, then, brings us to the next most logical form of surgical treatment of type III pancreatitis, and that is removal or destruction of the exocrine function of the pancreas. This may be done by total pancreatectomy, which is technically much more difficult for chronic pancreatitis, than for carcinoma, and also destroys the endocrine as well as the exocrine function of the pancreas.

Dr. Cannon's paper on pancreatic duct ligation with resultant atrophy of the acinar portion of the gland, with decreased exocrine function of the gland, seems to have considerable theoretic reasons to be worthy of further trial. This procedure has considerable experimental proof of pancreatic atrophy, as demonstrated by Ralli and Rubin; Nothman, Pratt and Benotti, Goldstein, Jacobsen, Telford and Roe, studies on blood amylase and lipase after duct ligation, and by histologic studies of Wang and Grossman.

Since experimentally it requires complete ductal ligation to produce acinar atrophy, and since Rienhoff and Pickrell in their anatomic studies of 250 cases of pancreatic duct relationship show 89 per cent with communications between the ducts of Wirsung and Santorini, it would seem mandatory to ligate both ducts, and Dr. Cannon's small series seem to substantiate this fact.

With the well known tendency of the pancreas spontaneously to re-fistulize following partial pancreatectomy as demonstrated by Whipple, I would like to ask Dr. Cannon why he has done only one duodenal content enzymatic activity study postoperatively on this group of cases?

Although Dr. Cannon's results are not too encouraging as yet, his series is still small; and it seems to me the procedure justifies further study for the treatment of a disease whose management to date has been so unsatisfactory.

JACK A. CANNON (closing): I wish to thank Dr. Paxton for his comments. I believe that his question regarding investigation of duodenal enzymes after surgery is a most pertinent one. We intend to do these studies on as many of the patients as we can but I would prefer to wait for a few months after operation before performing this procedure. The actual significance of the results may well be somewhat difficult to interpret, since I believe that enzymes can be present in the duodenum from bile succus entericus and saliva so that qualitative analysis of the enzymes themselves would probably be indicated.

Postmastectomy Lymphedema

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IN the past lack of accurate measurement of swelling of the arm following radical mastectomy has led to clinical misconceptions both of its incidence and severity. On the whole only the more severe grades of edema in the stoic patient were recorded, while minimal swelling in the arm of the hyper-reactive individual was often the source of much concern and bitter complaints. Any grading of edema on a subjective basis tends to be misleading because of the natural tendency to disregard cases with minimal swelling and to think only of those with functional impairment.

In order to have some rational basis for evaluation of postmastectomy swelling, measurements of both arms was undertaken in 100 normal women with no disease of the arm, shoulder girdle or chest. All measurements were made at a point 10 cm. below the acromion process in this control group as well as in patients after radical mastectomy, so that the area measured is comparable in all cases. The results obtained are summarized in Table 1. All data in this paper was calculated using the standard statistical methods after it had been demonstrated by so-called probit analysis that the results in the various groups of controls and radical mastectomy cases were not weighted. Our groups were found to be normally distributed about their means, so that a comparison of mean values gives a true indication of increase or decrease in arm size following various operative procedures.

From the data in Table 1 it is apparent that for moderate- to large-sized groups the mean arm size 10 cm. below the acromion is essentially the same in right and left arms for all practical purposes, and certainly well within the limits of one standard deviation. Using the "t" test of significance, the probability that the arm sizes were actually different was 0.50. To show significant difference between groups in biologic work the probability value must be at least in the range of 0.01–0.05 if not lower. If right and left arm size is essentially equal, then the mean values found in normals may be compared to the values found after radical mastectomy regardless of which side the lesion was on. Only 34 per cent of controls had a disparity in circumference between the arms of 0.75 cm. or greater, and only 9 per cent a disparity greater than 2 cm.

TABLE 1
ONE HUNDRED NORMAL CONTROLS*

	Right	Left
Mean	27.541 cm.	27.182 cm.
S.D.	3.33 cm.	3.31 cm.

* Equality of circumference, twenty-six patients; disparity of circumference, seventy-four patients.

Conclusions: (1) Mean of right arms is essentially equal to mean of left arms; (2) handedness has no correlation with edema.

Before reporting the results of our cases it is pertinent to offer some comment on axillary vein resection and postoperative irradiation as adjuncts to conventional radical mastectomy. As the senior author has previously reported,[4] primary resection of the axillary vein not only makes cases with adherent axillary nodes technically operable but also increases greatly the amount of lymphatic tissue which is removed. The so-called retrovenous pocket usually is not even seen at the time of conventional mastectomy. (Fig. 1.) Unlike the practice in some centers, there is no selectivity involved in our use of postoperative radiation in stage II cases. All patients with axillary nodal involvement are treated by x-radiation in a "tumor" dose of 3,000 to 3,500 r, h.v.l. 1.0 mm. Cu., to axillary and supraclavicular areas. Since all stage II cases are so treated, regardless of extent of axillary disease, there is thus no cause for confusion between edema due to extensive disease and edema due to radiation alone. Stage I cases were treated by radical mastectomy only.

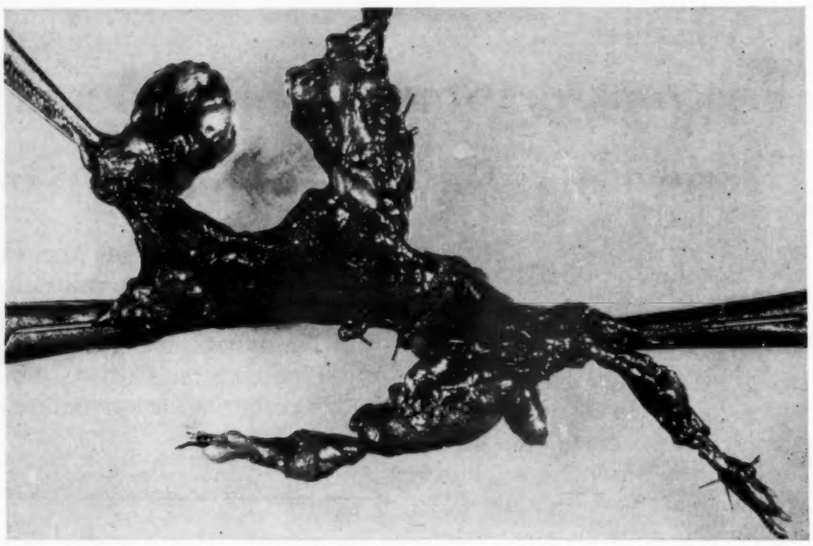

FIG. 1. Additional tissue dissected with resection of axillary vein after completion of conventional radical mastectomy.

Arm measurements 10 cm. below the acromion process were undertaken in our series of 143 private and clinic patients. Of these fifty-three had primary *en bloc* resection of the axillary vein, and eighty-two received postoperative radiation for stage II disease. This data fell naturally into four separate groups: (1) conventional radical mastectomy, forty-three patients; (2) radical mastectomy with axillary vein resection, seventeen patients; (3) conventional radical mastectomy with postoperative x-radiation, forty-six patients; and (4) radical mastectomy with axillary vein resection and postoperative x-radiation, thirty-six patients.

There are three main questions concerning postmastectomy swelling which we hope this data will help to resolve. (1) In what per cent of cases does swelling develop following conventional mastectomy? (2) How much additional swelling is added by vein resection and irradiation? (3) How important is the axillary vein in swelling?

In order to do this we have compared mean arm size after the conventional operation with that of the normal controls (29.586 cm. versus 27.54 cm.). The probability of these two mean values occurring in the same population is less than 1 in 1,000 ($p = 0.001$). Therefore significant swelling is present after conventional radical mastectomy. Seventy-two per cent show an increase in size greater than 0.75 cm. over the contralateral arm, and 24 per cent show 2 cm. or more of disparity. Thus conventional radical mastectomy of itself produces some swelling of the arm in 40 to 60 per cent of the patients operated upon. This agrees with the experience of other surgeons.[2,3]

We compared the populations shown in Table II in order to see what, if any, additional swelling postoperative irradiation and/or axillary vein resection added to conventional mastectomy.

The foregoing data indicates that vein resection produces no increase in postmastectomy swelling of the arm over conventional radical mastectomy. It further shows that irradiation does add to postmastectomy swelling. Since the only known method by which radiation causes swelling at any site is by lymphatic blockade, this is a rational assumption. Therefore, since vein resection or compression does not add and since x-radiation is a contributory factor, postmastectomy swelling of the arm is true lymphedema, as Halstead originally maintained thirty-five years ago. This idea was further investigated by means of axillary venograms to see if axillary compression without resection was correlated with edema. Ten patients were studied by axillary venograms using 70 per cent urokon sodium as the contrast medium. The Sandoz multiple cassette changer was used in order to have a number of films 0.1 second apart in time, rather than the single exposure venograms of the past. We wished to see the effect of the dye-blood flow relationship and what the degree of distention of the axillary vein at any one second would show.

All ten patients so investigated were nine months or more postoperative after conventional radical mastectomy; six had some degree of edema, four were without swelling. Only one of 6 patients with disparity in arm circumference greater than 1 cm. had evidence of axillary compression. Two of four patients with no edema had definite axillary compression. Multiple exposures in the patient with edema and axillary compression showed that the area of narrowing expanded to twice its apparent size with greater axillary distention; thus indicating that if only one exposure were done, a serious error in estimating the size of the narrowed area and its probable importance in edema might well be made. The conclusions drawn from this series of venograms is that axillary compression or narrowing is without effect on postoperative edema, at least in this small series. This is in contrast to the finding of

TABLE II

Type of Case	No. of Patients	Homolateral Arm		Contralateral Arm	
		Mean (cm.)	Variance*	Mean (cm.)	Variance*
Controls	100	Rt. arm 27.541	11.21	Lt. arm 27.182	10.914
Radical mastectomy	43	29.586	17.341	28.423	13.724
Radical mastectomy with radiation	46	31.941	20.627	30.091	15.030
Radical mastectomy with radiation and vein resectation	36	32.867	24.90	29.700	16.362
Radical mastectomy with vein resectation	17	30.141	24.315	28.847	21.850

* Variance = square of S.D. and is the number actually used in calculations comparing populations.

TABLE III
PROBABILITY VALUES*

Groups Compared	Homolateral Arms (P value)	Contralateral Arms (P value)	Significance
Standard radical mastectomy with standard radical mastectomy and radiation	0.01–0.02 probably different populations	0.05–0.1	Radical mastectomy increases edema
Standard radical mastectomy with radical mastectomy and vein resection	0.70 probably same population	0.9	Vein resection adds no edema
Radical mastectomy and radiation with radical mastectomy, radiation and vein resection	0.50 probably same population	0.7	Vein resection adds no edema to radiated cases
Radical mastectomy and vein resection with radical mastectomy, radiation and vein resection	0.05	0.8	Radiation, not vein resection increases edema
Conventional radical mastectomy with control population	0.001	.....	.....

* Level of significance 0.05 or less (one chance in twenty of being in same population).

Veal[5] and may well represent the better evaluation of axillary compression by multiple x-ray exposures and possibly the effect of contrast agents which produce less vasospasm.

Early in 1954 one of us (I. M.) abandoned the time-honored occlusive pressure dressing, for which step no originality is claimed. In fact, it took some twenty years of unthinking devotion to precedent, before the observations of others produced a conviction that occlusive pressure dressings over any major wound produces an environment comparable to that provided in a culture plate by the bacteriologist. In addition to providing an ideal bacterial playground, the traditional bulky dressing keeps the skin flaps artificially moist and unhealthy; the customary soft rubber drains rarely provide adequate drainage and the external pressure frequently fails to keep the skin flaps in apposition with their new bed.

Our present management of mastectomy wounds is to provide drainage by means of a large Foley catheter placed in the lower axillary area through a lateral stab wound, with distention of the bulb for self-retention of the catheter. Constant suction is maintained by an electric pump for six to ten days, except for short intervals of disconnection to permit bathroom privileges. The patient is out of bed in a chair for increasing periods each day with the suction maintained. No dressings of any kind are applied. Within a few hours the incision is self-sealed, while the suction removes the exudate and keeps the skin flaps in excellent apposition to the chest wall. The same management has been successfully employed when skin grafting is required.

At this writing, only fourteen patients with this sort of postoperative management are three months or more postoperative, for evaluation of wound healing and edema of the arm. Of these fourteen women ten enjoyed primary healing; in four healing was delayed by minor areas of wound separation and in none was there major necrosis and prolonged healing. Nine of the fourteen have edema of the arm on the operative side of 0.75 cm. or less, while only 2 present swelling of 2 cm. or more. Both the wound healing and the lack of edema in this small and statistically insignificant group are so favorable as to indicate the possibility of a gratifying reduction in postmastectomy edema with elimination of dressings and the use of constant suction.

CONCLUSIONS

1. A standard of reference for estimation of postmastectomy edema has been provided by determining the disparity of arm measurements in a control population of 100 "normal" women.

2. The degree of edema has been accurately determined in 143 postmastectomy patients, in fifty-three of whom resection of the axillary vein was done. Eighty-two patients had postoperative x-radiation.

3. In 72 per cent of the patients there was edema to the extent of 0.75 cm. or more disparity in arm circumference, and in 24 per cent 2 cm. or more.

4. Postoperative irradiation increases the degree of edema.

5. Resection of the axillary vein does not increase the degree of edema alone or in combination with postoperative irradiation.

6. Postmastectomy swelling is due to lymphatic obstruction and is a lymphedema.

7. Serial venograms in postmastectomy patients with and without edema fail to demonstrate any significant abnormalities in the axillary vein or its tributaries.

8. Preliminary results in the immediate postoperative management of fourteen patients by omission of all dressings and constant suction-drainage indicate the possibility of significant increase in primary healing and reduction of lymphedema.

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DISCUSSION

Gordon K. Smith (Los Angeles, Calif.): Dr. Macdonald has shown that fibrosis of tissues in the axilla leads to postmastectomy lymphedema. The prevention of fluid accumulation beneath skin flaps and prevention of marginal necrosis of the skin edges is of prime importance.

The efficacy of suction drainage by means of a catheter placed through a stab wound is so striking

that one wonders why it has only recently been added to our armamentarium. By this means, the flaps remain adherent to the chest wall and additional drains are unnecessary.

Marginal skin necrosis can best be prevented by relieving tension on the suture line with a few retention sutures placed through the skin and intercostal muscles. This decreases the length of the fulcrum between the medial and lateral aspects of the skin flaps and the suture line and prevents movement with respiration. Marginal skin necrosis additionally can be caused by infection of the sutured flaps.

I am thoroughly in accord with the principle and practice of leaving large occlusive pressure dressings off wounds. This concept is based on sound physiologic principles. It has been shown that cultures of wounds taken during operations and immediately following closure show them to be extensively contaminated with bacteria, predominately staphylococci. The source of these bacteria is from the skin of the patient, the air in the room, or from an infected area brought in contact with the wound. It is obvious that bacteria which are present in all wounds following closure are usually destroyed by the body's defenses provided that conditions suitable for their reproduction and multiplication are not present. Actually, in their effort to adjust metabolically to a new environment, bacteria are thwarted by what happens on an exposed surface. The exudate of a wound dries and is subjected to daylight and to an environment that is generally cooler than body temperature. This is injurious to bacterial reproduction, but drying is undoubtedly most injurious of all. This exudate is thus rapidly converted into a medium which inhibits bacterial growth instead of becoming an ideal culture medium as it frequently does in the moist, warm environment beneath the dressing.

Since 1949, I have omitted the conventional, time-honored, gauze dressings from clean wounds, covering only those which are drained through the incision. My experience with mastectomy wounds, although not as extensive as Dr. Macdonald's, has been as gratifying. Wounds are dry and clean and there is complete absence of folliculitis from adhesive tape. Some patients object to exposure of wounds on an esthetic basis. Over 100 patients, who had previously had surgery and were treated by conventional dressings, had their subsequent surgical incisions treated by exposure. They were enthusiastic about their comfort in the postoperative phase and the manner in which their wounds healed. It is my firm conviction that infection is introduced into a wound at the time of surgery and that if conditions are present which do not lend to bacterial multiplication, infection is not likely to develop.

In closing, I should like to state that I agree thoroughly with the principles that Dr. Macdonald has outlined. The prevention of serum accumulation under the skin flaps, by constant suction, and exposure of the wound, are fundamental principles which will lead to a much higher incidence of primary healing and, therefore, to a decrease in postmastectomy lymphedema.

W. Kenneth Jennings (Santa Barbara, Calif.): I want to call attention to an interesting complication which may develop in the swollen arm after mastectomy for carcinoma. I refer to lymphangiosarcoma. No doubt many of you are familiar with the report of Stewart and Treves which appeared in *Cancer* in January, 1948, and in which six cases of postmastectomy lymphangiosarcoma were documented. To date, a total of sixteen cases have appeared in the literature. Characteristically, the lesion makes its appearance after a long interval of freedom from cancer. The shortest interval between mastectomy and the onset of lymphangiosarcoma in any of the published cases was six years. It also is unique in that it is seen only in the lymphedematous extremity after mastectomy for cancer and not from lymphedema under any other circumstances. At least there have been no such cases reported. Typically, the tumor appears first as a mildly tender cutaneous nodule, purplish in color and usually on the anterior surface of the upper arm. Satellite nodules appear early and may coalesce or become widely disseminated.

Last fall we saw such a lesion in a sixty-two year old woman upon whom we had performed radical mastectomy for carcinoma six years earlier. She came in complaining of purplish nodules on the upper arm which she had attributed to a bruise. Biopsy section revealed a lymphangiosarcoma. Completely undifferentiated, closely spaced elongated spindle-shaped cells having poorly defined cell boundaries, a scant amount of cytoplasm and fairly large ovoid vesicular nuclei were noted. A few mitotic figures were present. In the central cellular portion a few irregular spaces filled with erythrocytes occurred. The diagnosis may be confused with hemangiosarcoma. These tumors are highly malignant and, to date, only one five-year survival has been reported. In our patient the lesion was widely removed and the incision carried down to include the underlying fascia; the defect was corrected by a full-thickness skin graft placed upon it.

I thought it might be timely to point out this rare and interesting complication of postmastectomy lymphangioma.

Alson Kilgore (San Francisco, Calif.): I can add only an expression of admiration for the carefully thought out and beautifully presented work of Dr. Macdonald and the delightful biological fantasies of Dr. Smith.

It does seem worth calling special attention, with enthusiastic concurrence, to the fact that they have not abandoned radical mastectomy as a method of attack upon cancer of the breast.

IAN MACDONALD (closing): I am grateful for the interesting commentary offered by the discussants. My debt to Gordon Smith is considerable, for he did much to convince me of the evils of occlusive dressings. He now suggests, I think with reason, that even an antibiotic ointment on a clean wound is undesirable obeisance to custom.

In respect to other points brought up, I have not yet seen a postmastectomy lymphangiosarcoma. It is of interest that Slaughter and Southwick recently reported a patient in whom the neoplasm was controlled by irradiation; perhaps amputation is not necessary.

We prefer sliding flaps to free grafts for closure, using the latter less than one in ten times.

We are using constant suction in all major wounds, particularly neck and groin dissections. In the latter, improvement in healing has been phenomenal. We use Chaffin-Pratt electric suction units.

I agree entirely that radical mastectomy still constitutes the best treatment for operable carcinoma of the breast, although I believe in rather stringent criteria of operability. American radiologists do not use the intensive dosage levels employed by McWhirter, from which the hazards of late tissue necrosis are not inconsiderable.

Varied incisions depending on the location of the primary lesion seem preferable. Usually I use a Greenough or transverse incisions in preference to the Halsted-Meyer type.

For determination of postmastectomy lymphedema, we have used an arbitrary minimum period of nine months. Only the few cases with no-dressing-suction management referred to herein are reported in terms of three months or more. Lymphedema seldom increases after six months.

Management of Acute Popliteal Arterial Occlusion

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Since the original application of sympathetic interruption for the treatment of peripheral vascular disease by Jaboulay[1] in 1899 there have been many modifications of surgical technic and clinical indications for this procedure. Today, however, a difference of opinion remains as to the proper role, extent and expected results of lumbar sympathectomy for the treatment of acute occlusion of a peripheral artery. The purpose of this report is to describe certain laboratory tests which assist in selecting patients with acute popliteal artery occlusion who will benefit from lumbar sympathectomy. The tests employed make it possible to locate accurately the site of arterial obstruction, determine the amount of arterial insufficiency and estimate the amount of functioning collateral circulation. Selected aspects of the anatomy, physiology and surgical technic of lumbar sympathectomy are reviewed. The problems of embolectomy and the use of anticoagulants are not discussed.

METHODS AND MATERIALS

The laboratory methods employed in studying these problems have been discussed in detail in previous reports.[2–4] They will be considered only briefly in this report. The pulsations of the extremities were measured with a recording oscillometer which was part of an electronic pneumoplethysmograph. The systolic blood pressures of the extremities were recorded using an electronic pneumoplethysmograph.[3] The general method employed for this measurement is shown in Figure 1. A plethysmographic cup is placed at the toe and is connected to the plethysmograph. In Figure 1 a meter is shown in place of the plethysmograph. The systolic blood pressure is measured by inflating a cuff to above systolic pressure then deflating the cuff until the plethysmographic stylus or meter needle suddenly moves, indicating that swelling of the part has occurred. This takes place when arterial blood leaks under the occluding cuff but cannot escape because of occlusion of the veins by the cuff. The pressure in the cuff at this time is the systolic blood pressure at this location. Peripheral nerve blocks were carried out by injecting 2 per cent procaine hydrochloride (novacain) or 1 per cent lidocaine hydrochloride (xylocaine) into the posterior tibial nerve just inferior to the internal malleolus or into the deep peroneal nerve between the great and second toe. (Fig. 2.) Skin temperatures were measured with a recording thermistor thermometer.[4] The venous congestion test of the foot was carried out with an occluding

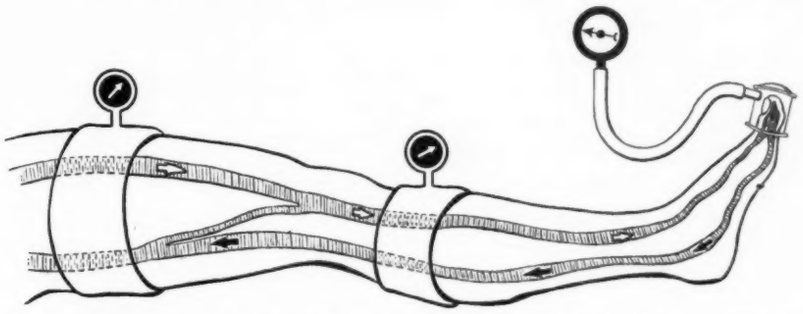

Fig. 1. Plethysmographic method for measuring systolic blood pressure of an extremity for calculating blood pressure indices.

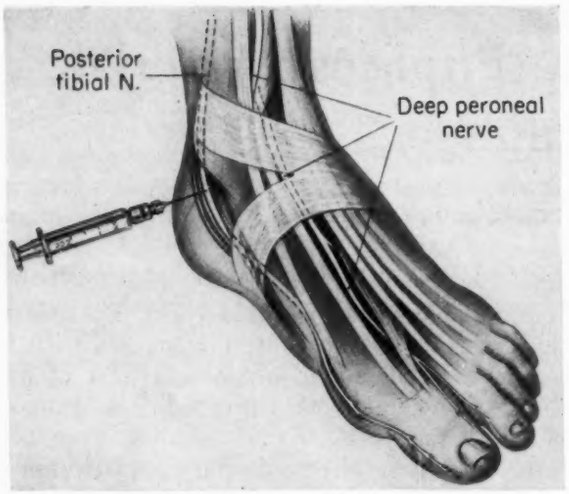

FIG. 2. Posterior tibial nerve block removed sympathetic vasoconstrictive impulse to the toe mediated through the posterior tibial nerve but not the deep peroneal nerve.

venous cuff at the ankle and a sensing cuff around the foot, the volume changes being recorded with the plethysmograph. The venous congestion test of the toe was performed using four venous occlusion cuffs at the ankles and were recorded with the plethysmograph. The effect of indirect body cooling was measured by recording skin temperatures before and after exposing the body to a room temperature of 16°C. for thirty minutes. All other measurements were recorded at a room temperature of 25° ± 1°C. Eight patients with acute popliteal arterial occlusion and thirty additional patients with arteriosclerosis obliterans without arterial emboli were studied.

RESULTS

All eight patients studied presented a clinical picture of acute popliteal embolus and all showed laboratory evidence of vasospasm of the peripheral blood vessels which could be released to a variable extent by a diagnostic posterior tibial nerve block. All patients had a limited lumbar sympathectomy (L3) done as an emergency. In all patients there was laboratory and clinical evidence of improved circulation to the foot and leg. In none was further surgery performed. All patients were returned to useful occupation. The details of a representative case is as follows:

S. R., a forty-eight year old man, was awakened by sudden pain in his right calf. He was seen six hours later at which time the right leg was pale and cold from the knee down. He was unable to stand because of motor weakness. Agonizing, constant and unrelenting pain was present at rest. The superficial veins were collapsed. The calf and foot were extremely tender. Four observers recorded no palpable pulsations in the right popliteal, posterior tibial or dorsalis pedis arteries. A clinical diagnosis of right popliteal artery occlusion by embolus was made. Laboratory findings were as follows:

TABLE 1

SYSTOLIC BLOOD PRESSURE

	BEFORE SYMPATHECTOMY		AFTER SYMPATHECTOMY	
	RIGHT	LEFT	RIGHT	LEFT
Above Elbow		140		130
Wrist		120		120
Above Knee	145	140	170	160
Below Knee	80	135	160	
Ankle	70	130	140	140
Index: Knee / Elbow	1.03	1.00	1.31	1.23
Ankle / Wrist	0.58	1.08	1.17	1.17

1. There was a definite reduction in the amplitude of the pulsations below the right knee and at the ankle while the pulsations above the knee were essentially normal. (Fig. 3.) The ankle/wrist pulsation index was 0.40 on the right as compared with 0.95 on the left. These findings suggested the presence of an abnormality in the region of the right popliteal artery.

2. The presence of collateral circulation around the obstruction was demonstrated as follows: A blood pressure cuff was placed above the knee and inflated above the systolic blood pressure (180 mm. of mercury) and sustained for five minutes. At the end of this time, the cuff was released. There was a definite flush or reactive hyperemia which was evident from the lower edge of the cuff to 2 inches below the knee. From this response, it was believed that the obstruction was low in the popliteal artery and that blood had reached the skin through collateral channels which had been dilated by this procedure.[5]

3. The systolic blood pressure was somewhat elevated above the right knee and lowered below the knee and at the ankle. (Table 1.)

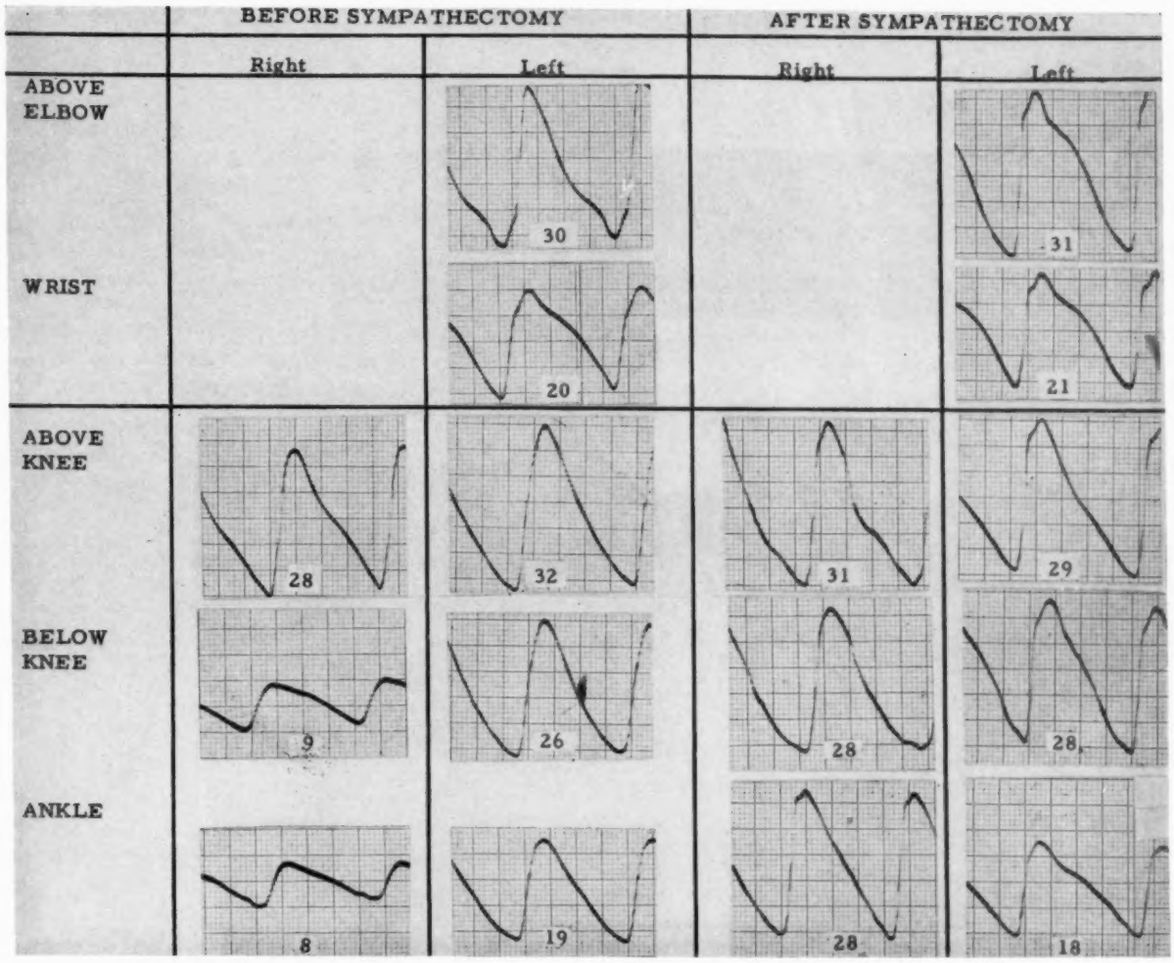

FIG. 3. Pulsations of extremities recorded by plethysmograph before and after sympathectomy.

This finding is characteristic of an obstruction in the region of the right popliteal artery.

4. Skin temperatures of the toes were measured before and after a posterior tibial nerve block to determine the probable outcome of a sympathectomy. (Fig. 4.) The temperature of the right second toe was 30.0 degrees before the block and 34.5 degrees after the block. These findings indicate the presence of a significant amount of vasoconstriction mediated through the sympathetic vasoconstrictor nerves and suggested that an appropriate lumbar sympathectomy would result in an increased blood flow to the toes.

5. The venous congestion test on the right foot was carried out before and after the posterior tibial nerve block. (Fig. 5.) The rate of swelling of the foot increased nine times following this procedure. This finding was interpreted as indicative of the presence of a strong neurogenic vasoconstrictive element limiting blood flow which could be interrupted by sympathectomy.

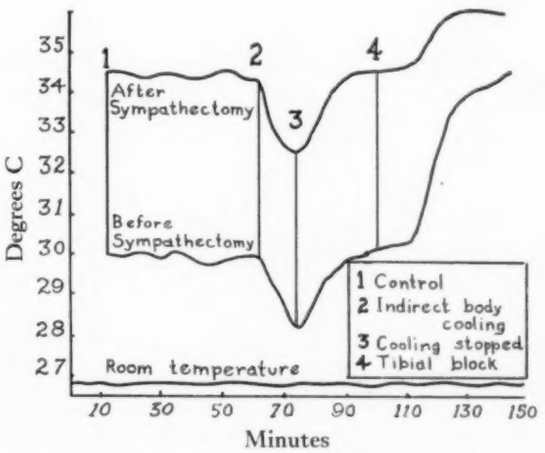

FIG. 4. Skin temperatures with body cooling and posterior tibial nerve block before and after limited (L3) lumbar sympathectomy.

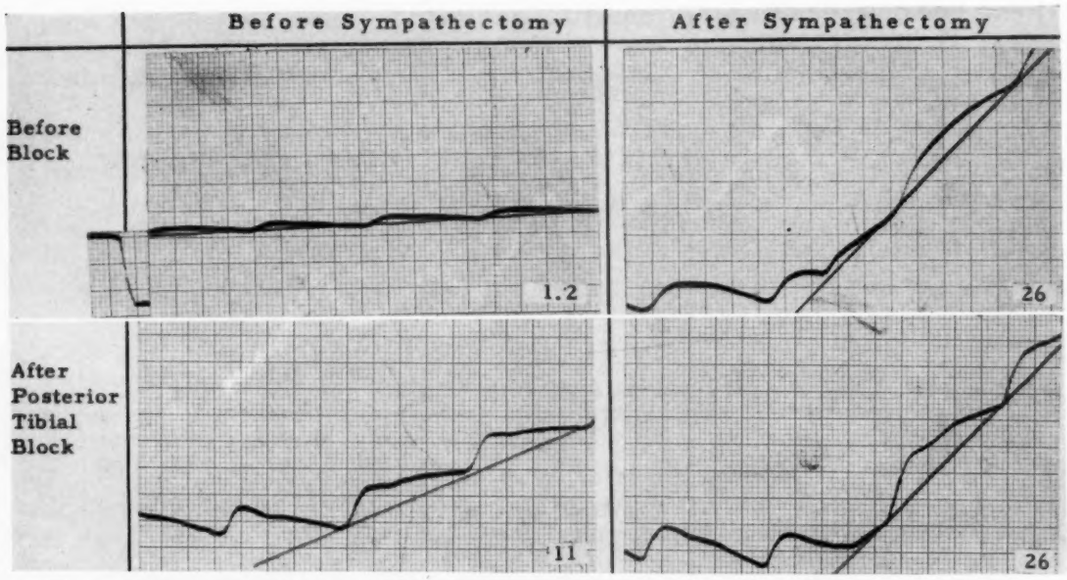

FIG. 5. Venous congestion test of foot; represents volume flow in cubic millimeters per second.

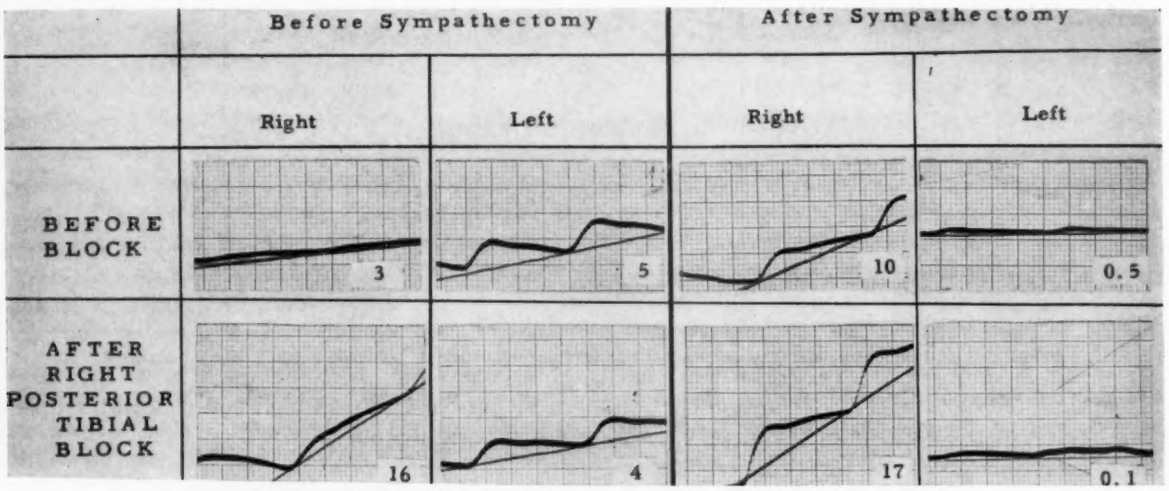

FIG. 6. Pulsations and blood flow of toes before and after sympathectomy.

6. A similar type of venous congestion test was carried out with the recording cup on the second toe. (Fig. 6.) The rate of swelling of the toe was greater than five times faster after the posterior tibial nerve block than before. This indicated likewise the presence of sympathetic vasoconstrictor activity released by the block.

In view of these findings, a limited lumbar sympathectomy was performed as an emergency. The operation performed in this case and in seven other cases of acute popliteal artery occlusion is a modification of the extraperitoneal approach to the lumbar sympathetic chain and ganglia which has been described by several surgeons.[6–10] The patient was placed in a supine position on an operating table which can be tilted in any desired direction. No special position, mechanical lifts or sandbags were required. Spinal anesthesia was used because of the excellent relaxation produced. However, care was taken to guard against a fall in blood pressure. In the "poor risk" patient, if spinal anesthesia is contraindicated, light general anesthesia may be combined with local infiltration of procaine hydrochloride (novacain) or lidocaine hydrochloride (xylocaine). A transverse incision was made just above the level of the umbilicus. (Fig. 7A.) From the margin of the rectus muscle, the incision was extended laterally for approximately 10 cm. The muscles of the abdominal wall were separated in the direction of their

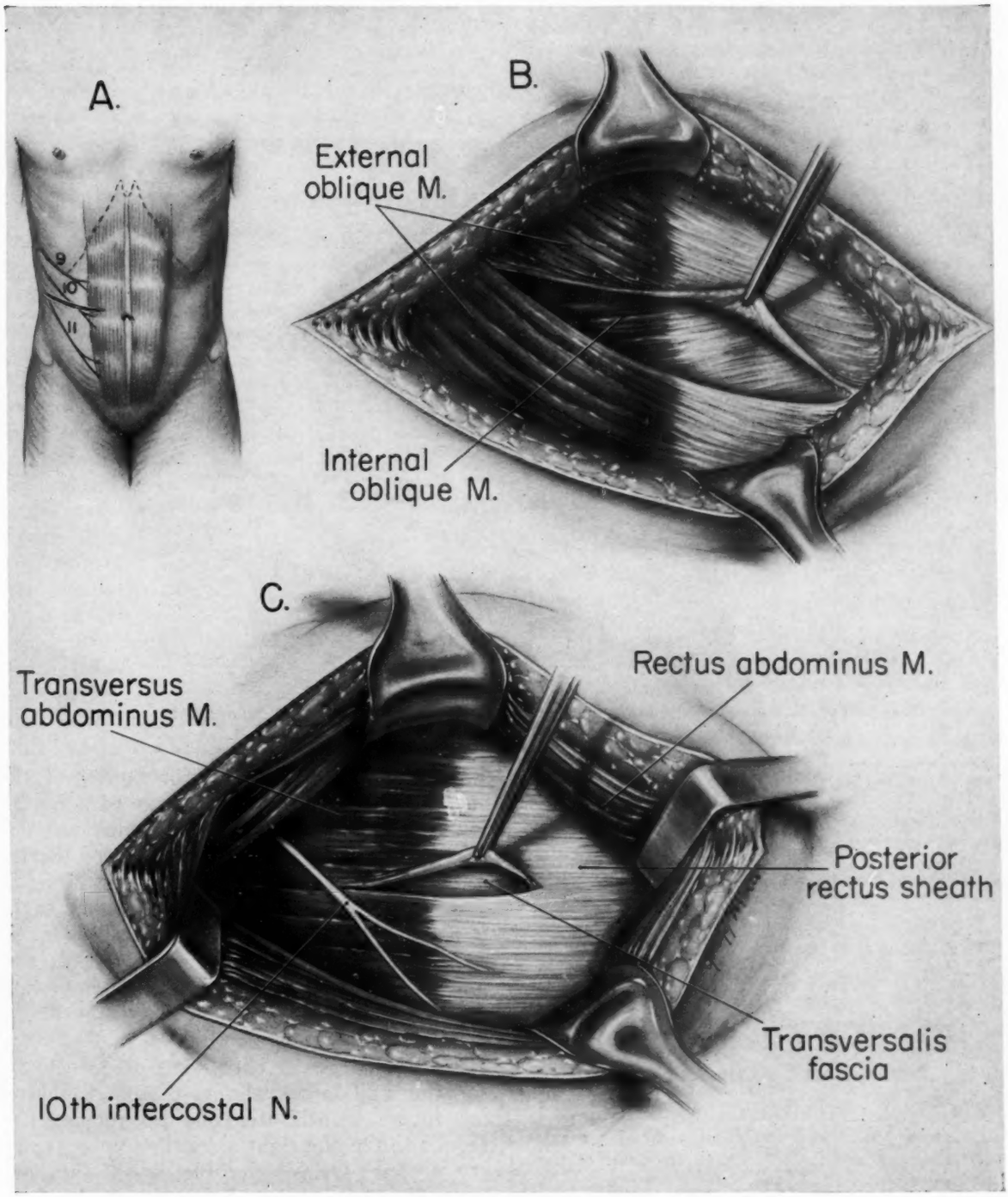

FIG. 7. Lumbar sympathectomy technic. A, incision; B, muscle splitting; C, tenth intercostal nerve. Division posterior rectus sheath.

fibers. (Fig. 7B.) The tenth nerve lying in the neurovascular plane between the transversus abdominus and the internal oblique was carefully retracted to avoid injury. (Fig. 7C.) Starting in the lateral gutter, the transversalis fascia and peritoneum were dissected free and retracted to the opposite side. When the medial border of the psoas muscle was reached, the table was tilted to aid exposure. At this point, the posterior rectus sheath, excluding the transversalis fascia and peritoneum, was split so that even better exposure of the sym-

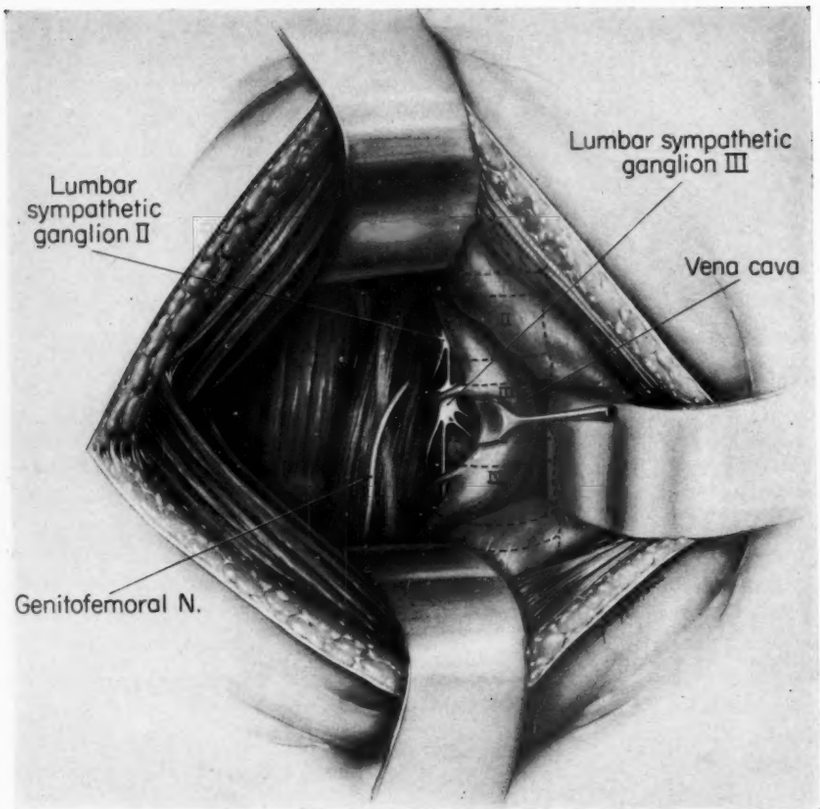

FIG. 8. Exposure lumbar paravertebral ganglionated sympathetic chain; note genitofemoral nerve.

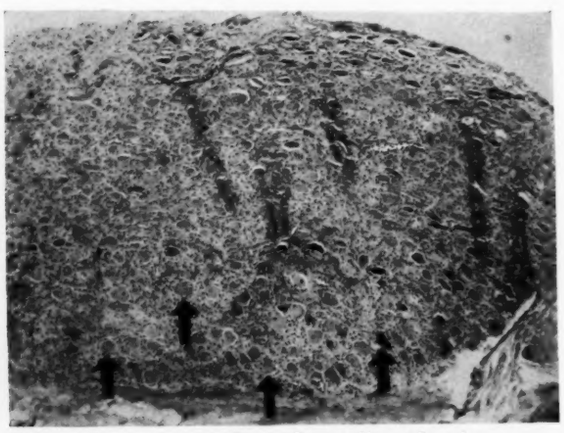

FIG. 9. Low power view of ganglion showing many nerve fibers and cells; arrows point to some of the ganglionic cells.

pathetic chain was obtained. (Fig. 7C.) The genitofemoral nerve lying on the medial border of the psoas muscle was protected from trauma to avoid a postoperative neuritis which has been a moderately severe complication and not as transient as some authors have reported. The sympathetic chain and a single ganglion lying between the superior border of the third lumbar vertebra and the inferior border of the fourth lumbar vertebra was removed. (Fig. 8.) This provided for an adequate, although not complete, sympathetic denervation of the leg and foot. Because of the known variations in the anatomy of the lumbar sympathetic chain and its ganglia, the surgical specimen so removed may include only one or possibly two ganglia. These represent the third or second and third lumbar ganglia. In this instance, one ganglion was removed (Fig. 9) and it was probably L3. Silver clips were placed at the superior and inferior limits of the dissection for future identification by roentgenogram. (Fig. 10.) On the right side, the lumbar veins can produce troublesome bleeding if they are torn. Frequently the chain will lie beneath these veins and has to be teased out from under them. After hemostatis was secured, the abdominal contents were allowed to fall back into their normal position. The wound was closed in layers using interrupted non-absorbable sutures. An occasional patient with postoperative abdominal distention required nasogastric suction for relief. The day of

ambulation depends upon the adequacy of the restored circulation. Recovery was uneventful.

At the end of a week, laboratory examinations were made to determine the degree of success obtained with this operation. The amplitude of the pulsations in the right leg had improved markedly. (Fig. 3.) The ankle/wrist index on the right was 1.33 as compared to a preoperative index of 0.40. The index on the right was actually greater than that on the left, which was 0.86. The systolic blood pressure at the right ankle was 140 mm. Hg as compared with 70 mm. Hg preoperatively. The ankle/wrist index was 1.17 as compared with 0.58 before sympathectomy. (Table 1.) The venous congestion test at the foot after sympathectomy showed considerable increase in peripheral circulation. (Fig. 5.) There was a volume increase to 26.0 cu. mm. per second after surgery as compared with 1.2 cu. mm. per second before sympathectomy. The venous congestion test applied to the right second toe after sympathectomy was 10.0 as compared to 3.0 before sympathectomy. (Fig. 6.) This graphically demonstrated a satisfactory improvement in circulation to the part as a result of the sympathectomy. The skin temperature of the second toe on the sympathectomized side was 34.5 degrees after sympathectomy as compared with 30.0 degrees before sympathectomy. (Fig. 4.)

The following tests for the completeness of sympathectomy were carried out. Indirect body cooling resulted in cooling of the limb, and a posterior tibial block resulted in moderate increase in skin temperature and improved blood flow of the toes. (Fig. 4.) The inspiratory reflex test was done before and after the posterior tibial nerve block. Before the block, the toe became smaller on taking a deep breath (sympathetic vasoconstrictive reflex), and after the block this reaction did not take place.

FIG. 10. Postoperative roentgenogram demonstrating silver clips at superior and inferior limits of excision of the right lumbar sympathetic chain and ganglion.

(Fig. 11.) These findings indicated that some vasoconstrictor fibers within the trunk of the posterior tibial nerve were still present.

In order to clarify the significance of some of these findings, certain studies were carried out in thirty additional patients. Effects of alcohol, alcohol and posterior tibial nerve block and sympathectomy on the skin temperatures of the second toe were studied in ten patients with arteriosclerosis obliterans. (Table 11.) This study was done to determine the advisability of giving alcohol by mouth, at the same time as performing a diagnostic posterior tibial nerve block as a vasodilating technic for

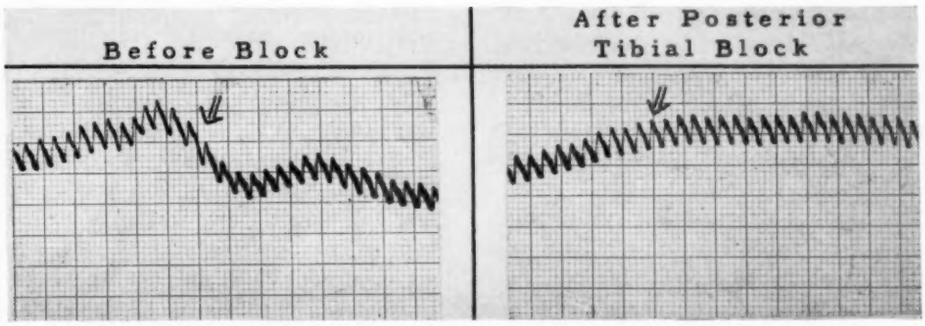

FIG. 11. Inspiratory reflex after sympathectomy. Arrow indicates inspiration.

determining the probable outcome of a lumbar sympathectomy. When alcohol alone was employed (2 ounces of undiluted whiskey), the average skin temperature of the second toes reached 33.3°C. as compared with alcohol and block which averaged 35.3°C. This value compared favorably with the results obtained 3.9 months after conventional lumbar sympathectomy (L2 and L3) the skin temperatures averaging 35.4°C.

TABLE II

EFFECT OF ALCOHOL, ALCOHOL AND BLOCK AND SYMPATHECTOMY ON SKIN TEMPERATURES

	SKIN TEMPERATURES (DEGREE C)				
	CONTROL	ALCOHOL	ALCOHOL AND BLOCK	LUMBAR SYMPATHECTOMY	MONTHS AFTER SYMPATHECTOMY
	24.2	34.0	35.0	35.5	8
	23.0	33.0	35.0	35.8	7
	25.2	35.0	35.2	35.0	1
	26.0	27.0	35.5	35.9	5
	27.1	35.0	35.6	35.8	3
	24.0	36.0	36.0	36.0	3
	24.5	34.0	35.5	35.8	3
	23.8	35.0	35.8	35.9	2
	27.0	34.0	35.5	33.0	4
	29.0	30.0	34.0	35.7	3
Mean	25.4	33.3	35.3	35.4	3.9
Min.	23.0	27.0	34.0	33.0	1.0
Max.	29.0	36.0	36.0	36.0	8.0

The amount of vasodilatation produced by a posterior tibial nerve block was studied in three patients. After the nerve block, the average skin temperature of the dorsum of the second toe was 35.0°C. The deep peroneal nerve was then blocked between the large and second toe and the temperature increased one-half degree to 35.5°C. Thus a posterior tibial nerve block removed a large part, but not all, of the sympathetic vasoconstrictive fibers going to the second toe.

The effect of a posterior tibial nerve block in ten patients after sympathectomy is shown in Table III. This indicates that only a very slight increase in circulation occurs after this procedure.

The effect of alcohol after lumbar sympathectomy was measured to determine if this agent acted centrally or peripherally. (Table IV.) Ten patients with moderate arteriosclerosis obliterans who had the conventional L2 and L3 sympathectomies two to six months previously were selected. All were tested for the inspiratory reflex and had posterior tibial nerve block and ganglionic blocking drugs to determine if the sympathectomy was complete or if regrowth of nerves or return of sympathetic function had taken place. In none of the patients had this occurred. The control skin temperature of the second toe averaged 34.2°C. After 2 ounces of whiskey it averaged 35.1°C. or an increase of 1.0°C. Thus it was concluded that alcohol had, in part at least, a direct vasodilating effect upon the arterial system.

COMMENTS

Following an acute occlusion of a peripheral artery, it is not always possible by clinical observation alone to locate accurately the site of the obstruction, to determine the degree of arterial insufficiency, estimate the amount of functioning collateral circulation or select accurately patients who will benefit by lumbar sympathectomy. A standard battery of objective tests were employed as an adjunct to clinical observations. By the proper application and interpretation of the results of these tests one can select those patients with acute occlusion of the popliteal artery who will benefit by an emergency lumbar sympathectomy. With these tests the results of sympathectomy can be predicted with some accuracy, thereby

clarifying the indications for the operation. The indiscriminate application of sympathectomy in all cases of acute arterial occlusion would lead to poor results in patients in whom the vasospastic element is minimal or absent.

The various laboratory tests which were employed in this study were selected to give information which help determine the probable outcome of a low lumbar sympathectomy if it were performed. The pulsations and systolic blood pressure gradients of the extremities assisted in locating the obstruction in the arterial tree and helped to differentiate rigid from obstructed vessels. Rigid vessels with little obstruction produced low pulsations and high pressures while obstructed arteries resulted in a lowering of both of these measurements. The venous congestion test of the toe and foot gave values which were related to blood flow of the part in cu. mm./second and these measurements made before and after a diagnostic nerve block gave information relative to the potential blood flow to the diseased extremity. The skin temperature of the toes gave evidence of the amount of skin circulation and the potential skin circulation could be estimated by making this measurement before and after the posterior tibial nerve block. The completeness of the sympathectomy was evaluated by recording skin temperatures, pulsations of the digits and blood flow after posterior tibial nerve block, medical ganglionic blockade, inspiration and indirect body heating and cooling.

TABLE III

POSTERIOR TIBIAL NERVE BLOCK AFTER SYMPATHECTOMY

Patient Number	Months After Sympathectomy	Skin Temperatures		Difference
		Before	After	
1	10	34.0	35.0	1.0
2	1	35.1	35.5	0.4
3	3	35.5	35.6	0.1
4	3	35.0	35.4	0.4
5	3	35.8	35.8	0.0
6	4	35.2	35.3	0.1
7	3	34.0	34.1	0.1
8	3	34.9	35.0	0.1
9	4	35.1	35.1	0.0
10	3	35.2	35.3	0.1
Mean		34.9	35.2	0.2
Minimum		34.0	34.1	0.0
Maximum		35.8	35.8	1.0

The clinical diagnosis of acute arterial occlusion is rarely difficult. Albright and Leonard[12] summarized the clinical findings of arterial occlusion as the five "P's"—pain, pallor, paralysis, pulselessness and paresthesia. If these are present the diagnosis is established. When the palpable peripheral pulses are questionable, the more sensitive plethysmographic technic of obtaining and recording the pulses, blood flow and systolic pressures are of value.

TABLE IV

EFFECT OF ALCOHOL AFTER LUMBAR SYMPATHECTOMY ON SKIN TEMPERATURES OF TOE

	SKIN TEMPERATURE (DEGREE C)		
	CONTROL	ALCOHOL	DIFFERENCE
	35.0	35.8	0.8
	34.2	35.3	1.1
	34.0	35.1	1.1
	35.0	35.7	0.7
	33.9	34.3	1.4
	34.1	35.5	1.4
	33.8	34.9	1.1
	34.0	34.1	1.1
	34.1	35.0	0.9
	34.3	35.3	1.0
Mean	34.2	35.1	1.0
Min.	33.8	34.1	0.7
Max.	35.0	35.8	1.4

It is of interest that the case presented demonstrated clinically these five characteristic diagnostic findings; however, additional valuable information was obtained by the laboratory examinations. For example, they revealed clearly the presence of pulses, collateral circulation and a significant blood pressure in the leg. It is believed that laboratory examination is of definite importance in acute occlusion of an artery as there is usually intense vasospasm associated with this condition. If the spasm is of neurogenic origin it can be released by interrupting the sympathetic impulses to the involved toes by a posterior tibial nerve block. This is far easier to do than a lumbar sympathetic block and the results are probably more reliable as an indication for sympathectomy.

A limited lumbar sympathectomy (L3) was selected for these patients in an effort to produce an increase in circulation below the site of the obstruction so that shunting of blood proximal to the obstruction would not occur. For clarification of this concept a brief review of the anatomy and physiology of the sympathetic nerve supply to the peripheral blood vessels is offered. (Fig. 12.)

The efferent sympathetic pathways to the peripheral vascular system of the lower extremities can be divided into three sets of neurons.[10,13,14] The primary neurons probably have their origin in the central ganglia of the diencephalon with an indeterminate amount of cortical control. These neurons establish synapses with the cells in the intermediolateral column of the spinal cord. The secondary neurons (preganglionic fibers) begin in the intermediolateral column of the cord, and extend to the paravertebral ganglionated sympathetic chain through the white rami communicantes. The tertiary neurons (postganglionic fibers) arise in the sympathetic ganglia and through the gray rami rejoin the spinal nerves to supply the blood vessels in the lower extremities. Considerable overlapping occurs in the preganglionic connections and some in the intermediate ganglia and spinal cord.[13–16] It is generally agreed that the lowest preganglionic connection is usually at the level of the second but may be as low as the third spinal root.[7,10,11,13–16] Therefore, for all practical purposes, sympathectomy for vascular deficiency in the foot and leg will be sufficient if the chain and outflow from the third spinal root alone are removed. It is not necessary to remove the first or fourth lumbar ganglia. For the treatment of acute popliteal artery occlusion a low thoracic-high lumbar sympathectomy is not indicated because vascular shunts in the thigh are opened up and the foot will actually be deprived of blood. This has been observed many times clinically. Perhaps this is the basis for the reluctance of some to do a sympathectomy on a patient with an acute arterial occlusion. As demonstrated in this report, excision of L3 produced a satisfactory sympathectomy even though it was not complete. Holf et al.[17] are in agreement with these principles. Some believe that removal of the postganglionic fibers, which have their origin from the fourth lumbar ganglion or below, should be avoided. However, after postganglionic denervation, the possibility of epinephrine sensitization has probably been overemphasized. Regeneration of the sympathetic nervous

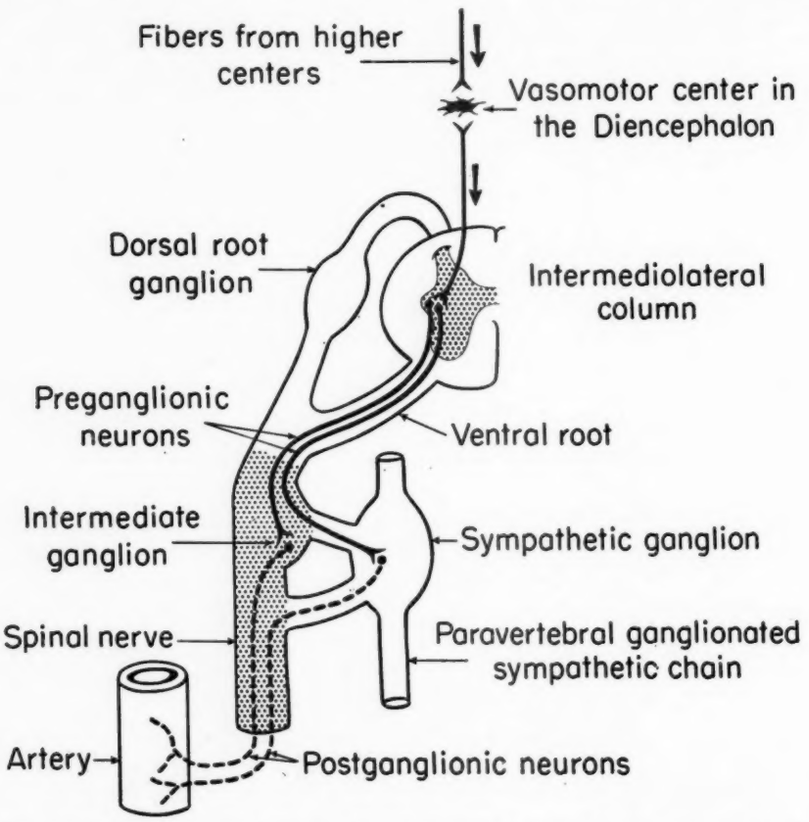

FIG. 12. Efferent sympathetic pathways to the peripheral vascular system of the lower extremities; note intermediate ganglion.

system after surgical sympathectomy has not been established in a large series of autopsied cases. In many instances what is thought to be regeneration probably represents overlapping of sympathetic nerve activity or failure to remove the few fibers which reach the periphery through the spinal nerves by synapsing in the intermediate ganglia without ever entering the paravertebral sympathetic ganglia. (Fig. 12.)

SUMMARY

Several objective laboratory tests have been described which assist in selecting patients with acute popliteal arterial obstruction for emergency lumbar sympathectomy. From information so obtained it is possible to locate the site of obstruction, estimate the amount of arterial insufficiency and determine the amount of collateral circulation. If it can be demonstrated that vasospasm is a major factor in the ischemia of the limb, lumbar sympathectomy should be done as an emergency procedure and not as a last resort. Limited lumbar sympathectomy (L3) as described produced satisfactory but incomplete sympathetic denervation of the toes in the patients evaluated by these procedures.

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Resection of Abdominal Arteriosclerotic Aneurysm

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ANEURYSMS of the aorta have challenged the ingenuity of surgeons for centuries. Recorded literature dates back to Cooper in 1817. Since that time many procedures have been tried and reported, with variable results.[1–5] Their success, however, had to be judged against a very uncertain knowledge of what the untreated course of these aneurysms would be. Statistical studies have been reported.[6–10] The usual conclusions pointed to a poor prognosis but there is not complete agreement on this point. Cranley et al.[9] reported thirty-two cases of arteriosclerotic aneurysm, with autopsy findings. In this group only 25 per cent of the patients died as a result of the aneurysm. Estes,[10] however, reported that of the sixty-nine patients followed 63 per cent succumbed to the effects of the aneurysm. Furthermore, most statistics have dealt largely with leutic aneurysms, lesions which are now rare as a result of medical progress.

Excision of the aneurysm and replacement by a graft was suggested by Carrel[11] in 1910, but it was not until 1952 that Dubost[12] reported the first successful resection of an aneurysm with replacement by a homograft. In 1953 DeBakey and Cooley[13] reported a series of twelve cases with successful treatment by resection and replacement by a graft. A very lucid description of the technic utilized and some of the difficulties encountered were given. In subsequent papers[14,15] they reported a larger series of aortic resections which were performed for aneurysm or occlusive disease. Seven deaths occurred in a series of twenty-six resections performed for abdominal aneurysm, the survivors being free of complications. More recently, several papers[16,17] dealing with the use of synthetic prostheses for replacement have been published. The results to date are comparable with those in which homografts were used. The advantages of easy and inexpensive procurement and storage, as well as accurate fit of the prosthesis, are immediately apparent.

The author has had experience with ten cases of arteriosclerotic abdominal aortic aneurysm treated by resection and replacement with homografts. Four of the grafts used were stored in a balanced electrolyte solution at 1°C. and the others were freeze dried. In this series of ten cases three deaths and several major complications occurred. The purpose of this paper is to report the technical problems, complications and autopsy findings in these cases.

The patients were selected without a crystallized scheme. Indication for surgery was the presence of an aneurysm in a patient who, after careful evaluation, was considered likely to withstand the procedure. No patient was refused surgery if the symptoms attributed to the aneurysm were severe. The three deaths which occurred were in relatively poor-risk individuals. Each of the patients had severe hypertension and the rapid and severe progression of symptoms suggested a grave prognosis. During the fifteen months of this study, three patients with aneurysm were refused surgery because of advanced cardiovascular renal disease. Each had severe hypertension and moderate azotemia; one of the patients had had a cerebral vascular accident with incomplete recovery from hemiplegia. The symptoms attributed to the aneurysms in these patients were mild and posed no problems *per se*. All three of these patients are still living and are in about the same state of health four, ten and thirteen months since the diagnosis of aortic aneurysm.

Three patients were in moderately serious condition at the time of surgery because of recent hemorrhage from the aneurysm or definite palpable enlargement. There was a severe inflammatory reaction about these aneurysms, together with lymphadenopathy

and edema, which made dissection a little more tedious and sanguine. Two of these patients died.

Aortography by the translumbar technic was utilized in seven cases. Although it offered additional information in some cases, it certainly was not indispensable. No reactions or complications were attributed to this procedure. A flat plate of the abdomen was of value in confirming the diagnosis of aneurysm, as the aneu-

TABLE 1*

Case	Age and Sex	Race	Blood Pressure	Period of Occlusion (min.)	Method of Graft Storage	Date of Operation	Results	Remarks
1, G. E. L.	55, M	W	140/80	65	Bal. elect. 13 days	1-14-54	Excellent	Diarrhea postoperatively associated with rectal stricture; at celiotomy aorta normal to palpation
2, R. B. H.	67, M	W	160/90	220	Bal. elect. 16 days	1-22-54	Excellent	Mild paresthesias of legs for 1 wk.; diarrhea postoperatively
3, H. V. M.	54, M	W	240/140	75	Bal. elect. 9 days	2-24-54	Deceased 14th postoperative day	Embolectomy right popliteal artery postoperatively; diarrhea postoperatively; death due to septicemia and hemorrhage from right renal artery
4, E. H. C.	59, M	W	220/110	60	Bal. elect. 6 days	4-2-54	Deceased 36 hr. postoperatively	Ascending thrombosis beginning in legs; shock; renal infarcts; graft patent
5, C. C. M.	69, M	W	160/90	42	Freeze dried	4-14-54	Excellent	Diarrhea postoperatively; embolectomy right femoral artery
6, F. A. B.	51, M	W	150/90	55	Freeze dried	5-10-54	Excellent	Mild diarrhea postoperatively; rapid enlargement of mass immediately preoperatively
7, L. S.	56, M	W	140/82	75	Freeze dried	8-5-54	Excellent	Benign postoperative course
8, E. C.	64, M	W	260/140	105	Freeze dried	9-9-54	Deceased 10th postoperative day	Exsanguination from superior suture line; renal infarcts
9, T. K.	67, F	W	150/90	105	Freeze dried	10-7-54	Excellent	Right renal artery clamped for 80 min.; no residual damage; mild postoperative diarrhea
10, J. H.	69, M	W	145/92	220	Freeze dried	10-20-54	Poor	Left iliac artery occluded postoperatively; ischemic neuritis—residual paralysis; benign rectal stricture; severe postoperative diarrhea

* Etiology: all were arteriosclerotic aneurysms.

rysmal wall was usually sufficiently calcified to be outlined.

Table 1 outlines the pertinent data on the cases being reported. Cases 3, 4 and 8 will be reported in more detail, with inclusion of the autopsy findings. It is to be noted that the patients who died had significant hypertension. However, the cause of death was different in each case.

TECHNICAL CONSIDERATIONS

Both spinal and general anesthesia have been used and no preference has been established. It is believed that keeping the operating table flat or the patient's feet slightly down, is helpful in protecting the patient's legs during the ischemic period. The feet should be protected from the weight of drapes or other possible pressure points. The legs should not be used for intravenous infusions.

A long midline incision is used and a self-retaining retractor is placed for exposure. The posterior parietal peritoneum is incised upward to the ligament of Treitz, and the small bowel is packed upward and to the right. Additional exposure may be obtained by mobilizing the right colon and enclosing the small bowel and colon in a Lahey bag.

Dissection of the aneurysm and iliac vessels should progress in a cleavage plane as close to the artery as possible. The dissection should first be directed toward the superior end of the aneurysm and the relatively normal aorta immediately proximal to it. After the artery is separated sufficiently, umbilical tape is passed about it for emergency use. The inferior mesenteric artery is ligated and divided close to its origin and the aneurysm is freed on the sides and inferiorly as far as possible without retracting or otherwise disturbing it. When manipulation is necessary, the aorta above and the iliac arteries below should be clamped to reduce the possibility of freeing emboli from the thrombus within. Immediately before the clamps are closed, 15 to 20 mg. of heparin are injected into each iliac artery to counteract the thrombophilic effect of stagnation. Failure to use heparin in this manner was directly responsible for the complications leading to the death of one of the patients in the series (Case 4). DeBakey advises that 10 mg. be injected directly into the aneurysm. It is believed that this may not protect adequately; no difficulties have resulted from the larger doses. The aortic clamp should be applied as high as possible but care should be taken not to encroach upon the renal arteries. Encroachment upon the renal vessel required nephrectomy in one case but caused no difficulty in another. The iliac arteries are then divided and mobilization of the aneurysm is completed. This part of the procedure is facilitated by forward traction on the aneurysm and serial clamping of the lumbar arteries as they are encountered. Freeing the aorta from the adjacent vena cava and iliac veins is the most difficult, tedious and hazardous part of the entire dissection. It is believed that hyaluronidase applied on small dissecting sponges has been helpful at this stage. If necessary, the adventitia of the aorta may be left on the vena cava. Any accidental rent in the vena cava should be sutured without attempting to clamp and ligate.

When the aneurysm is mobilized, it is removed by transecting the aorta at a level immediately superior to it. A cuff of at least 4 to 5 mm. should remain below the clamp to facilitate suture. The aorta should be transected as squarely and cleanly as possible. It has been found helpful to do a minimal thromboendarterectomy on the accessible aortic cuff and on the iliac segments. This allows the needle to pass through the wall easily without the danger of elevating thick, calcified, loosely adherent intimal plaques that could embolize.[18] In addition, the aorta is made more pliable and can be reefed a little to make it fit the usually smaller graft more accurately. (Fig. 1.) The adventitia and media have adequate holding power. No difficulty has been encountered in these cases or in thirty thromboendarterectomies in which the same layers were used for closing the vessels.

The graft which has been soaking in 0.9 normal saline solution during the dissection, and which an assistant has been preparing by tying and suturing small branches, is now ready for anastomosis. Linton[19] has stated that one should not attempt to perform the operation if only a single graft is available and that it is preferable to make a selection from several grafts. There is complete agreement with this statement. The end of the graft can be beveled slightly to make up for the discrepancy in size. It should not be beveled sharply, nor should a longitudinal incision be made to lengthen the edge of the graft in order to make it match the host aorta. (Fig. 2.) There is danger of delayed

disruption of the suture line when these methods are used. (Case 7.)[20]

The proximal anastomosis is made first, using a simple through and through continuous suture of No. 0000 arterial silk. The stitches should be placed about 2 mm. apart and about

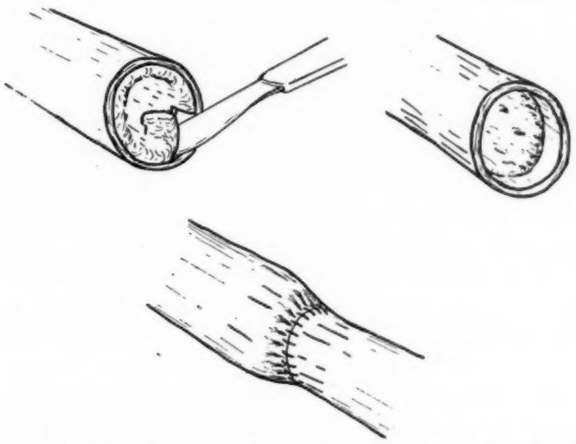

FIG. 1. Diagrammatically depicts thromboendarterectomy of the available aortic cuff, allowing the aorta to be reefed sufficiently to fit the graft.

3 mm. deep, and should be pulled up snugly. A continuous suture without interruption can be used without danger of a purse-string effect. The anastomosis is inspected and any obvious defects are closed with interrupted sutures. The graft is cut short enough to fit with very slight tension as it will elongate when blood flow is established and may kink sufficiently to obliterate one of the iliac arteries if it is too long.[18] One of the lower anastomoses is made using the same technic. This iliac artery is then temporarily clamped off at its origin from the aorta. The clamp occluding the aorta is slowly opened allowing 20 to 30 ml. of blood to flush out the still open iliac artery, after which it is clamped off and the clamp on the completed iliac artery removed thereby re-establishing blood flow to that lower extremity. This maneuver is intended to protect against embolization of thrombi that may have formed during occlusion of the aorta. The remaining anastomosis is completed and all clamps are removed, re-establishing normal circulation. Bleeding from the suture lines can be controlled by light pressure on a hemostatic sponge. Further attempts to suture are usually inadvisable. During each anastomosis the suture line and adjacent vessel are irrigated with saline solution containing 1 mg. of heparin per milliliter to protect against incipient thrombosis. In this group of cases the aorta was clamped from 42 to 200 minutes without apparent harm.

When the surgeon is satisfied with the integrity of the anastomoses, the peritoneum is carefully closed over the graft. The relatively

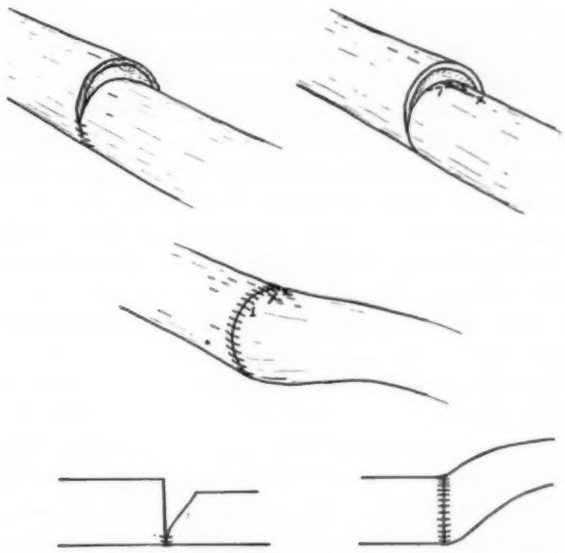

FIG. 2. Diagrammatically depicts two methods used to help make up for discrepancy in size. The longitudinal incision is possibly dangerous and not advised. Sharp beveling of the graft is also potentially hazardous.

rapid and firm adherence of the peritoneum and fatty tissue to the graft probably helps to prevent delayed leakage. This assumption is made on the basis of the celiotomy and autopsy findings.

Sympathectomy is performed only if there is preoperative evidence of arterial insufficiency. The rationale of prophylactic sympathectomy in patients with adequate arterial supply is questionable since vasospasm has not been a problem.

Postoperative management offers no problems peculiar to the procedure and early ambulation is practiced. Heparin is not given unless it is specifically indicated.

COMPLICATIONS

Embolic occlusion of the right common femoral artery occurred in Case 5, and of the right popliteal artery in Case 3. These emboli originated from the aneurysm and were freed during surgery. They were not diagnosed in either case for several hours postoperatively,

at which time the patients were immediately returned to the operating room and embolectomy performed under local anesthesia. Return of distal pulsation was prompt and permanent, although one patient (Case 3) expired on the seventeenth postoperative day.

Thrombosis of the left common iliac artery with severe ischemia of the leg occurred immediately following the operation in Case 10 resulting in ischemic neuritis and complete paralysis of the extremity. The patient's very poor condition precluded re-exploration early enough to expect satisfactory results. The extremity remains viable. A subsequent aortogram disclosed a long and tortuous graft with an angulation obstruction of the left common iliac artery. This was confirmed at celiotomy.

The left renal artery was occluded accidently for eighty-two minutes in Case 9. Following removal of the clamp good pulsations occurred. The kidney was not disturbed. Excretory urograms three months postoperatively disclosed good bilateral renal function. In Case 10 a clamp damaged the left renal artery necessitating nephrectomy. Good right renal function was known to have existed at the time.

Diarrhea beginning in the early postoperative period has been aggravating and not fully explained. In Case 10 it led to cachexia which required vigorous supportive therapy. Only Case 7 escaped postoperative diarrhea. In two of the patients a diaphragmatic type of rectal stricture was present at about 9 cm. from the anal orifice. Biopsy was negative for carcinoma in both cases, as was the Frei test. Moore[18] reported a similar stricture in one of his cases. This co-existence of benign rectal stricture and aortic aneurysm may be coincidental or causal relationship may exist.

No complications occurred after the second postoperative week. The seven surviving patients are free of difficulty, with the exception of one patient (Case 10) who has residual ischemic neuritis.

Death due to septicemia with miliary abscesses, renal failure and retroperitoneal hemorrhage from rupture of the right renal artery occurred on the seventeenth postoperative day in Case 3. The graft and anastomoses were intact and free of thrombus. The entire right kidney was infarcted and numerous small infarcts were present in the left kidney. Microscopic examination indicated that these small infarcts could have originated postoperatively. The peritoneum was tightly adherent to the graft and was difficult to separate. Although there is doubt as to whether it would resist expansion, it was believed it would help prevent small leaks, especially from the suture line. The elastic fibers of the graft were well preserved. The graft was lined with a thin layer of fibrous tissue. The difficulties leading to death in this patient were probably not preventable. It is possible, but not likely, that septicemia could have been introduced by a contaminated graft, even though culture studies on the graft were negative. The rupture of the right renal artery could have been initiated by injuries incident to the operation.

One patient (Case 4) died in shock thirty-six hours after operation. Death was attributed to thrombosis of the extremities that propagated proximally to involve the external iliac arteries. The graft was patent. There was an estimated 400 cc. of blood in the retroperitoneal space. Both kidneys showed very recent small infarcts, more numerous in the left. Failure to heparinize the extremities for the first hour of occlusion is believed responsible for the thrombosis leading to death. This was preventable.

Another patient (Case 8) died on the tenth postoperative day due to hemorrhage from the proximal suture line. There was massive hemoperitoneum. In this case the peritoneum had not completely reapproximated. A single stitch had cut through the graft forming a 2 mm. hole from which the patient became exsanguinated. The remainder of the suture lines were adequate. The peritoneum was firmly adherent to the graft. The kidneys showed infarcts which were judged to be ten days old, again worse on the left side. The remainder of the autopsy examination was not remarkable. The method of enlarging the graft circumference is believed responsible for the delayed leak. In addition it is believed that emergency exploration at the time bleeding was diagnosed would have probably saved this patient, as the leak was very small and could probably have been controlled.

In addition to the foregoing examinations, Cases 1 and 10 manifested rectal strictures for which celiotomy was performed four months later. The aorta was normal to palpation and the extent of the graft could not be ascertained. No attempt was made to expose the grafts as they were well covered with peritoneum and fat.

CASE REPORTS

CASE 3. H. V. M., a fifty-four year old white man, was admitted to the Long Beach Veterans Administration Hospital on January 29, 1954, complaining of severe, persistent, abdominal and low back pain of three weeks' duration. The patient had a five-year history of hypertension and for four years had had episodes of dyspnea and orthopnea. He had noted an irregular pulse for about one year. Medication consisted of digitalis 0.1 gm. daily and various antihypertensive drugs. There was a past history of pulmonary tuberculosis. Physical examination disclosed a large pulsating abdominal mass arising in the epigastrium and measuring 15 cm. in length and 12 cm. in width. It was tender to palpation. Blood pressure was 241/140 mm. Hg. All pedal pulses were present. Fundoscopic examination showed grade III hypertensive retinopathy. Laboratory findings included an electrocardiogram which showed frequent ventricular extrasystoles and left axis deviation. Roentgenogram of the chest showed the residuals of bilateral upper lobe tuberculosis with considerable upward hilar retraction. Blood urea nitrogen was 40 mg. per cent; other chemical tests and a hemogram were normal. A moderate number of leukocytes and granular casts were present in the urine which was otherwise normal. Serologic tests were negative. A roentgenogram of the abdomen showed a large aneurysm well outlined by calcium within the wall. An aortogram was done but gave little added information.

On February 24, 1954, under spinal anesthesia supplemented with pentothal,® N_2O_2 and O_2, the abdomen was explored through a long midline incision. No pathologic change other than the large aortic aneurysm was found. This extended from just below the renal arteries into the common iliac arteries but did not involve the iliac bifurcations. After it was completely mobilized and excised, it was replaced by a bifurcation homograft that had been stored for nine days in a balanced electrolyte solution of 1°C. The aorta was occluded for seventy-five minutes during the procedure. There was very little bleeding on removing the clamps and this was readily controlled by pressure with a hemostatic sponge. The peritoneum was closed over the graft. Sympathectomy was not performed.

Shortly after returning to his bed the patient complained of pain and coldness in his right leg. Examination disclosed a blanched right leg with the pedal pulses absent. The femoral and popliteal pulses were normal. The patient was immediately returned to the operating room and the popliteal artery was exposed under local procaine anesthesia. The embolus which was located just superior to the popliteal bifurcation was removed without difficulty and good back bleeding occurred. The arteriotomy wound was closed with a running through and through No. ooooo silk. The pedal pulses returned and remained good thereafter. The composition of the embolus grossly resembled that of the thrombus present in the aneurysm.

On the seventh postoperative day the patient had a severe chill and a spiking temperature of 104°F. He had been receiving penicillin 600,000 units daily. This was discontinued. A blood culture was obtained which was positive for Aerobacter aerogenes. Urine cultures showed the same organism. The organism, on test, was positive only to aureomycin. This was given in doses to 0.5 gm. four times a day but without effect. Repeated blood transfusions were given along with aureomycin intravenously without altering the course of the disease. The patient became jaundiced on the fourteenth postoperative day and on the fifteenth day ecchymoses were present with gingival bleeding. Platelet counts showed severe thrombocytopenia. Cortisone was given along with whole fresh blood transfusions. Fever, jaundice and bleeding continued, and the patient expired on the seventeenth postoperative day.

Autopsy disclosed a massive retroperitoneal hemorrhage arising from the right renal artery. The graft was intact and without evidence of thrombus. The entire right kidney was infarcted and there were numerous infarcts of the left kidney which were judged to be several weeks old. There was severe pyelonephritis and septicopyemia of the liver. The adrenals showed marked lipoid depletion of the cortex with hemorrhages and focal necrosis. Death was attributed to hemorrhage and septicemia.

CASE 4. E. C., a fifty-nine year old white man, was admitted to the hospital complaining of excruciating abdominal pain radiating into the back and down the legs. The pain had been present for one week during which time repeated injections of morphine were given without relief. A mass had been noted for about one year prior to hospitalization. The

patient had been treated for hypertension for several years.

Roentgenograms of the abdomen showed clear evidence of the aneurysm. Aortograms were not done. The electrocardiogram showed left ventricular hypertrophy.

Operation was performed on April 2, 1954, under general anesthesia. The aneurysm was large, extending from just below the renal arteries to the bifurcation of each common iliac artery. The aneurysm was mobilized with some difficulty and excised. Heparin was not injected into the iliac arteries prior to occlusion of the aorta due to an oversight. This was discovered one hour later at which time 20 mg. of heparin were injected into each external iliac artery. The reconstituted freeze-dried graft was sutured in place without difficulty. It was anastomosed to both internal and external iliac arteries. The peritoneum was closed over the graft after it was certain the graft was functioning. Sympathectomy was not done. Femoral pulses were good immediately postoperatively and the right popliteal pulse was palpable. The feet were cool and the pedal pulses absent. The patient complained of severe pain in his feet after he had fully recovered from the anesthesia. Heparin was started but in another twelve hours the pain had extended to the knees. The right popliteal pulse was no longer obtainable. Heavy analgesia was necessary to control the pain. In another twelve hours the femoral pulses were absent and the legs became cold and mottled. The patient went into shock and expired within an hour.

At autopsy there was an estimated 400 cc. of blood in the retroperitoneal space. The graft was intact and free of thrombus down to the bifurcation of the common iliacs. The external iliac arteries were both thrombosed as was the arterial tree from there distalward. The kidneys showed numerous infarcts, varying from 2 to 6 mm. in size, slightly more numerous on the left. The arteries generally were sclerotic, but there were no other findings pertinent to surgery.

Case 8. E. C., a sixty-four year old white man, was admitted to the Long Beach Veterans Administration Hospital on August 23, 1954, complaining of recurrent episodes of right subcostal pain of one year's duration. This had been extremely severe for the six days prior to admission. The patient had noted a tender pulsating abdominal mass for four days. He had been on hypertensive management for eight years.

Physical examination revealed a moderate sized, very tender, pulsating mass in the mid-abdominal region. Femoral and pedal pulses were normal. Blood pressure was 256/140 mm. Hg.

Roentgenograms of the abdomen disclosed an aneurysm with a partly calcified wall. An aortogram was done but contributed little. Electrocardiogram showed evidence of an old anterior septal infarct and left ventricular hypertrophy.

On September 9, 1954, under spinal anesthesia, the abdomen was opened through a long midline incision. The aneurysm was mobilized and resected from just below the renal arteries to 2 cm. below the bifurcation of the aorta. Immediately before clamping, 20 mg. of heparin was injected into each iliac artery. The resection was accomplished without difficulty. However, the aorta was considerably dilated and anastomosis of the reconstituted freeze-dried homograft was difficult because of the disparity in size. A 1 cm. longitudinal incision in the graft was made to increase its circumference. By this maneuver the anastomosis was satisfactorily completed. The graft functioned well. The peritoneum was not reapproximated over the graft.

On the first postoperative day the patient had good pedal pulses and was ambulatory. He did well until the eighth postoperative day when he fainted. Hemoglobin determination the next morning was 13 gm. per cent as compared to 14 gm. per cent two days previously. There were no abnormal abdominal findings and the patient had no complaints at this time. He fainted again on the evening of the ninth postoperative day and 500 cc. of blood was required to return the blood pressure to its usual level. The next morning the abdomen was slightly distended and peristalsis was hypoactive. Hemoglobin, hematocrit, pulse and blood pressure levels were followed for twelve hours and showed no fluctuation. Shortly after this the patient complained of severe abdominal pain and blood pressure and pulse were barely obtainable. He expired while 1,000 cc. of whole blood were being given under pressure.

Autopsy examination disclosed nearly 4,000 cc. of blood within the peritoneal cavity. The graft was patent and did not contain a thrombus. It was firmly covered by peritoneum in its

lower two-thirds; but the upper one-third including the anastomosis was bare. There was a 2 mm. hole in the suture line anteriorly where a suture had pulled through the graft. This was at the point where the longitudinal incision had been made. The aorta and the graft were tested for suture holding quality, and the graft cut through with very little tension while the endarterectomized aorta held very well. Microscopic examination of the graft showed a well preserved elastic coat but few other identifiable cells. The kidneys showed numerous small infarcts, more in the left than in the right. These were judged by microscopic examination to be about ten days old. The colon was normal throughout and showed no deleterious effects from ligation of the inferior mesenteric artery. The remainder of the examination was non-contributory.

SUMMARY

1. Ten cases of arteriosclerotic abdominal aortic aneurysm have been reported. There were three deaths in this series, one each from ascending thrombosis and shock, uncontrolled sepsis and uremia, and exsanguination from the proximal suture line.

2. Patients with advanced cardiovascular renal disease in whom the symptoms attributable to the aneurysm were mild were not offered surgery.

3. No patient was refused surgery regardless of his physical status if the symptoms were severe.

4. Protection against embolization and thrombosis by clamping and heparinization are important considerations in the operation.

5. A choice of grafts at the time of operation and sufficient time for their reconstitution is emphasized.

6. Partial thromboendarterectomy is advocated.

7. Continuous through and through silk suture is satisfactory. The graft should be short enough. Careful reperitonization may help prevent delayed leak.

8. Complications included peripheral emboli and thrombosis in two cases and faulty application of the aortic clamp occluding the left renal artery in another two cases.

9. Two patients had coexisting benign rectal strictures requiring later surgical excision. This occurrence appears to be more than coincidental.

10. Six of the patients are now free of symptoms. One patient has disabling residuals of ischemic neuritis.

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Follow-up Observation and Evaluation of Sympathectomies in Obliterative Arterial Disease

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THIS report represents one surgeon's experience and evaluation of the treatment in fifty-three cases, of five categories of obliterative arterial disease by lumbar sympathectomy. The categories to be considered are: (1) arteriosclerotic peripheral arterial circulatory insufficiency; (2) arteriosclerotic peripheral arterial insufficiency with preceding relatively acute arterial thrombosis (one week to three months); (3) Buerger's disease; (4) arteriosclerotic peripheral arterial insufficiency with associated popliteal aneurysm; and (5) diabetic endarteritis.

Postoperative evaluations of lumbar sympathectomy in similar patient groups have been excellently reported by Coller, DeBakey, Trimble, Smithwick, Freeman and their coworkers.[1–5] No attempt will be made to compare their reported end results accurately with this series. It can be stated that they are in general accord, with a few exceptions which will be pointed out later. The cases in this series have been drawn entirely from private practice and perhaps comprise a group of quite favorable material.

Until the observations of Reichert,[6] Atlas,[7] Smithwick[4] and others, beginning about 1938, it was the general teaching that lumbar sympathectomy was of little or no value in the treatment of obliterative arterial disease. This reasoning was predicated on the general lack of evidence of actual or seeming vasoconstriction in this type of peripheral circulatory insufficiency. It was assumed that pain of somatic origin had an exclusively central nervous system transport, and that only the pain of pure vasoconstrictive etiology could be expected to be aided by sympathetic denervation. The observations previously cited have demonstrated that this limitation does not exist, and that lumbar sympathectomy in properly selected cases of obliterative arterial disease is a reasonable therapeutic tool.

METHODS AND CASE SELECTION

The selection of the fifty-three cases to be analyzed was based on the usual items of history and physical examination: (1) claudication (rest pain, effort pain, night pain); (2) color changes (blanching extremity on elevation, cyanotic rubor in dependency); (3) presence or absence of pedal pulses; (4) trophic changes; (5) response to lumbar ganglionic block.

All cases were screened medically for contraindications to surgery of a systemic nature. The operative procedure was carried out through the usual anterolateral muscle-splitting approach. A bilateral procedure was done in one stage if the bilateral procedure seemed indicated. In those cases with high arterial obstruction with thigh and hip claudication the procedure was extended to include the first lumbar ganglion or higher. Trimble et al.[3] have stated that one-stage bilateral procedures should not be performed. This series shows no evidence that such a limitation exists. Preoperative lumbar ganglion diagnostic blocks were frequently utilized using pontocaine (0.15 per cent dilution) as the blocking agent. After skin temperature observations were made, the observer walked with the patient to determine the degree of improvement in effort tolerance. This procedure proved of progressively decreasing value in case selection. It frequently had merit, however, in demonstrating to a patient what might be expected as a postoperative result. Demonstration of a vasoconstrictive component was frequently not clearly seen even though subjective pain was relieved and effort tolerance increased. This raises obvious ques-

tions as to the mechanism of pain relief by sympathetic denervation.

PREOPERATIVE CASE APPRAISAL

I. Arteriosclerotic Peripheral Circulatory Insufficiency

There were thirty-six cases in this category, with an average age of sixty years and equivalent sex incidence (twenty men and sixteen women). Associated systemic diseases such as diabetes or coronary insufficiency was not of unusual frequency. All of these patients had claudication of varying degrees of severity. It will be noted that twelve of the thirty-six patients had pain at rest, twenty-two had night pain and fourteen had pain with the effort of walking one-half block at a slow gait. Only five had open terminal necrotic ulcerations. Four of the thirty-six patients had bilaterally absent femoral pulses with the subjective and objective findings of the Leriche syndrome. Not one instance of demonstrable segmental arterial obstruction was found despite aortographic studies on likely candidates. It is interesting that significant blanching on elevation of the legs was seen in thirty-two of thirty-six cases, while cyanotic rubor was recorded in only nineteen instances. Fifteen preoperative diagnostic blocks were done, with relief of pain and improvement in effort tolerance in thirteen of the group. Elevation of skin temperature did not parallel improvement in effort tolerance. The greatest increase in skin temperature (20°F.) occurred in a patient with marked arterial obliteration extending up the aorta to just below the level of the renal arteries. (Table I.)

II. Arteriosclerotic Peripheral Circulatory Insufficiency Associated with Relatively Recent Acute Arterial Thrombosis

The eight cases in this group were of interest in that a long-standing history of claudication was not present. Time intervals of one week to three months prior to seeking medical advice were the rule. Acute episodes of leg and foot pain, coldness and transient anesthesia were the histories of these cases. The average age (fifty-three years) was seven years less than the uncomplicated arteriosclerotic group. Six of the patients were men and two were women. Two of the eight patients were diabetics and none had any associated significant evidences of systemic cardiovascular disease. In all but one, minor trauma such as sitting in an automobile for long periods or squatting at work seemed to be the precipitating incident. Claudication was the presenting symptom, with rest pain in seven of eight cases, and four plus night pain in seven cases. In three the obstruction was as high as the femoral level. (Table II.)

TABLE I

PREOPERATIVE STATUS OF THIRTY-SIX PATIENTS WITH ARTERIOSCLEROTIC PERIPHERAL CIRCULATORY INSUFFICIENCY

Claudication (36):	
Pain at rest	12
Pain after ½ block	14
Pain after 1 block	8
Pain after 2 blocks	2
Numbness and tingling (8)	
Night pain (22):	
1 plus	3
2 plus	6
3 plus	4
4 plus	9
Pulses absent:	
Bilateral:	
Femoral	4
Popliteal	19
D. pedis	25
P. tibial	25
Unilateral:	
Femoral	7
Popliteal	8
D. pedis	11
P. tibial	6
Color changes:	
Blanching on elevation	32
Cyanotic rubor in dependency	19
Avascular terminal ulceration (6)	
Diagnostic preoperative lumbar ganglionic block (15):	
Relief of pain on effort with varying degrees of skin temperature elevation	13
No relief	2
(Following lumbar sympathectomy improved, 12; not improved, 3)	
Associated systemic disease:	
Mild antecedent cerebrovascular accident	1
Diabetes mellitus	4
Essential hypertension	6

III. Buerger's Disease

It is not surprising that only seven cases of this syndrome, of a severity warranting sympathectomy, were seen. Buerger's disease is not

common. Hamlin and co-workers[8] reported only 180 cases in a twenty-year span at the Massachusetts General Hospital. In establishing this diagnosis they apparently do not insist on the association of migrating phlebitis with peripheral arterial occlusion. All seven cases of this series met this requirement. The age span of twenty-four to fifty-five years is as expected; average age, forty years. All patients were men. Only two had evidences of finger involvement, and these were of minimal severity. Tobacco was an obsession in all but one, who was an abstinent, nicotine-free Scandinavian. Claudication and night pain was severe in all cases. One of four patients receiving a preoperative diagnostic lumbar ganglionic block had no relief of pain or increase of effort tolerance. It will be seen later in the follow-up appraisal that he had an excellent result from lumbar sympathectomy. (Table III.)

TABLE II

PREOPERATIVE STATUS OF EIGHT PATIENTS WITH ARTERIOSCLEROTIC PERIPHERAL CIRCULATORY INSUFFICIENCY ASSOCIATED WITH RELATIVELY RECENT ACUTE ARTERIAL THROMBOSIS

Claudication (8):	
Pain at rest	7
After 1 block	1
Night pain (7):	
4 plus	7
Pulses absent:	
Bilateral:	
Femoral	0
Popliteal	2
D. pedis	2
P. tibial	1
Unilateral:	
Femoral	3
Popliteal	6
D. pedis	8
P. tibial	6
Color changes:	
Blanching on elevation	8
Cyanotic rubor in dependency	4
Avascular terminal ulceration (2)	
Diagnostic preoperative lumbar ganglionic block (4):	
Relief of pain on effort with varying degrees of skin temperature elevation	3
No relief	1
(Following lumbar sympathectomy improved, 4)	
Associated systemic disease:	
Diabetes mellitus	2

IV. Arteriosclerotic Peripheral Arterial Insufficiency with Associated Popliteal Aneurysm

This relatively rare complication of arteriosclerotic disease was seen in only one case suitable for surgical correction after the fashion of Linton.[9] It is to be noted, however, that over the period of this case study popliteal aneurysm as the precipitating focus of acute arterial obstruction with gangrene was seen four times.

TABLE III

PREOPERATIVE STATUS OF SEVEN PATIENTS WITH BUERGER'S DISEASE

Claudication (7):	
Pain at rest	4
Pain after ½ block	1
Pain after 1 block	3
Numbness and tingling (3)	
Night pain (7):	
4 plus	7
Pulses absent:	
Bilateral:	
Femoral	0
Popliteal	4
D. pedis	7
P. tibial	7
Unilateral:	
Femoral	1
Popliteal	0
D. pedis	0
P. tibial	0
Color changes:	
Blanching on elevation	7
Cyanotic rubor in dependency	6
Avascular terminal ulceration (2)	
Diagnostic preoperative lumbar ganglionic block (4):	
Relief in pain on effort with varying degrees of skin temperature elevation	3
No relief	1
(Following lumbar sympathectomy improved, 4)	
Associated systemic disease	0

The patient, a fifty-five year old man, had only mild signs preoperatively of peripheral arterial insufficiency. In addition to a large, pulsating, expansile right popliteal mass he demonstrated slight blanching on elevation, cyanotic rubor in dependency and claudication followed walking one block at a brisk pace.

V. Diabetic Endarteritis

This solitary case was obviously poorly selected. The patient was a seventy-one year

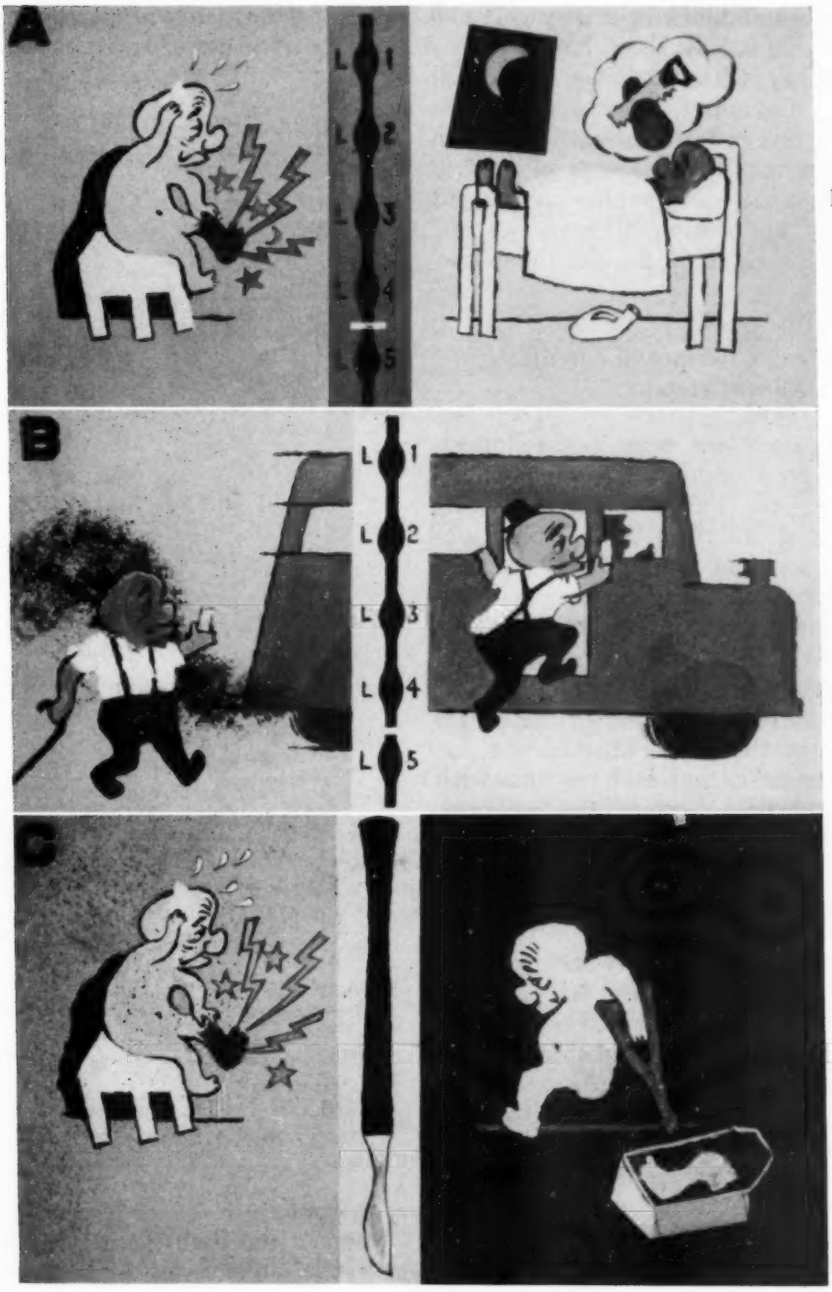

FIG. 1.

old white diabetic man with marked claudication, rest pain, blanching on elevation, and numbness and tingling in his toes. The pedal pulses were, however, palpable. Strangely, he responded well in a disarming way to a preoperative diagnostic block.

END RESULTS

The classification of end results in the standard descriptive terms of excellent, good, fair and poor is, in this group of cases, quite meaningless. It would aid considerably if there were a visual type of communication equally recognizable to the patient, the surgeon and to the surgeon's colleagues. (Fig. 1A to C.)

The patient with pain at rest with leg dependent, who has spent three weeks trying to sleep in a chair and after lumbar sympathectomy can sleep painlessly supine, might consider his end result "excellent." Another, who

has limped after one block at a slow gait and postoperatively can walk three blocks slowly without limp might consider his end result "fair to good." Certainly the patient who has no relief of pain and undergoes low thigh amputation can be placed in the pain-wracked "poor" category. His condition has pointed out the limitations or possible dangers[7] of the procedure. This, then, is the basis of the following appraisal.

I. Arteriosclerotic Peripheral Circulatory Insufficiency

These thirty-six cases were assessed postoperatively over a minimum period of six months to one year, and a maximum of six years. Exceptions are three poor results, obviously permanent, in the immediate postoperative period. One-third of the group fell into the minimal follow-up period, while the remaining two-thirds were examined at intervals of two to six years postoperatively.

It will be seen from the accompanying charts that by and large the relief obtained was significant. Of interest is that four patients with high arterial (aortic) obstruction have been rehabilitated. This suggests that perhaps a preliminary bilateral lumbar sympathectomy followed by a period of observation not less than eight months should precede the infinitely more hazardous endarterectomy in high segmental obstructions. Trimble et al.[3] pointed out that not infrequently limited immediate relief was followed by progressive slow increase in effort tolerance. In this series this phenomenon, possibly explained by the experimental work of Reichert,[6] was also noted. The patient with rest pain with leg dependent, in the absence of terminal necrotic ulceration, was invariably relieved of his distress immediately. In the weeks and months that followed, effort tolerance on walking was extended.

Patients with end results falling into the category of "poor," with one exception, had open terminal avascular ulcerations at the time of surgery. The occasional success in this type of case reported by Coller et al.[1] and DeBakey et al.[2] was not enjoyed. One case classified as "good" end result had returned to work in Alaska as a construction engineer for three years after lumbar sympathectomy. A fall with mild trauma to the leg resulted in gangrene and subsequent amputation. (Table IV.)

II. Arteriosclerotic Peripheral Arterial Insufficiency Associated with Relatively Recent Acute Arterial Thrombosis

In this group of eight cases was the only postoperative death in the total of fifty-three cases.

TABLE IV

END RESULT APPRAISAL OF THIRTY-SIX PATIENTS WITH ARTERIOSCLEROTIC PERIPHERAL ARTERIAL INSUFFICIENCY

	Follow-up	
Time	Relief	No Relief
6 yr.	2	..
5 yr.	2	..
4 yr.	10	..
3 yr.	5	..
2 yr.	2	1
6 mo.–1 yr.	9	1
8 mo.	..	1
3 mo.	..	1
1 mo.	..	1
2 wk.	..	1

	Results	
Status	Immediately P.O.	End Result
Improved	29	30
Excellent	19	22
Fair to good	10	8
Not improved	7	6

Miscellaneous	
Operative mortality	0
Subsequent amputations (5 poor end results)	6
Deaths subsequent to surgery (3):	
Coronary thrombosis (1 yr. p.o. poor end result with amputation; 6 yr. and 6½ yr. p.o. in cases classified as excellent)	3
Subsequent non-fatal associated disease:	
Coronary thrombosis	5
Cerebrovascular accidents (all without residual disability)	4
Unilateral lumbar sympathectomy	12
Bilateral lumbar sympathectomy	24

This patient was a sixty-three year old diabetic woman who died unexpectedly and suddenly on the seventh postoperative day of acute coronary occlusion.

The cases were followed up over periods ranging from six months to six and a half years. Five of the surviving seven patients enjoyed a consistently excellent result. (Table V.)

III. Buerger's Disease

The seven cases followed up over periods ranging from three to seven years have presented no untoward problems except the amputations of first toes on three occasions in two tobacco slaves. (Table VI.)

IV. Arteriosclerotic Peripheral Circulatory Insufficiency with Associated Popliteal Aneurysm

An uncomplicated convalescence in the single case, after a preliminary lumbar sympathectomy followed by Linton's excision of the aneurysm without evidence of circulatory embarrassment, would seem to establish the rightness of Linton's approach.

TABLE V

END RESULT APPRAISAL OF EIGHT PATIENTS WITH ARTERIOSCLEROTIC PERIPHERAL ARTERIAL INSUFFICIENCY ASSOCIATED WITH RELATIVELY RECENT ACUTE ARTERIAL THROMBOSIS

Time	*Follow-up* Relief	No Relief
6½ yr.	1	..
5 yr.	1	..
4 yr.	2	..
3 yr.	1	..
1 yr.	..	1
6 mo.	..	1

Status	*Results* Immediately P.O.	End Result
Improved	5	..
Excellent	3	3
Fair to good	2	3*
Not improved	2	1

Miscellaneous

Operative mortality (63 yr. old diabetic woman died 7th postoperative day of acute coronary occlusion)	1
Subsequent amputations	1
Deaths subsequent to surgery (died 1½ yr. p.o. of acute coronary thrombosis; low thigh amputation 3 mo. p.o.	1
Subsequent non-fatal associated disease (2):	
Coronary thrombosis (1½ yr. p.o.)	1
Cerebrovascular accident without residue (2 yr. p.o.)	1
Unilateral lumbar sympathectomy	8
Bilateral lumbar sympathectomy	0

* One case with poor result immediately following surgery; good result at end of 6 mo. with complete relief of pain at rest and good improvement of effort tolerance.

V. Diabetic Endarteritis

Despite the encouraging response to a preoperative lumbar ganglionic block this patient had no relief of pain, was further burdened by a persistent burning pain in both thighs and died six months postoperatively of an acute coronary occlusion.

In regard to this still unexplained phenomenon of deep-seated burning pain in the thighs following lumbar sympathectomy, it occurred in ten of the fifty-three aforementioned cases. Coller et al.[1] describe this as a posterior and lateral "neuralgia" in the thighs, lasting only seven days, and attribute it to operative trauma. This was not the case in the foregoing series. The pain was deep in the thigh as subjectively described, burning in character, of varying severity and occasionally associated with hyperesthesia of the skin of the anterior thigh. In no instance was it disabling; in all it was annoying. The distress as described invariably lasted at least four months, and in one instance one year. Analgesics such as empirin were adequate to render this burning distress tolerable.

TABLE VI

END RESULT APPRAISAL OF SEVEN PATIENTS WITH BUERGER'S DISEASE

Time (yr.)	*Follow-up* Relief	No Relief
7	2	0
5	1	0
4	3	0
3	1	0

Status	*Results* Immediately P.O.	End Result
Improved	7	7
Excellent	6	6
Fair to good	1	1
Not improved	0	0

Miscellaneous

Mortality at or following surgery	0
Subsequent amputations	2
1st toe bilateral	
1st toe unilateral	
Subsequent non-fatal associated disease (1):	
Coronary thrombosis	1
Unilateral lumbar sympathectomy	3
Bilateral lumbar sympathectomy	4

CASE REPORTS

CASE 1. W. C., a fifty-five year old man, was first seen at the Swedish Hospital, Seattle, on February 25, 1948. He had a history of severe intermittent claudication for one year. For the preceding four months he had right leg calf and foot pain at rest, numbness and tingling in the right foot, and had noted increasing reddish purple discoloration of the foot in the dependent position.

During the month prior to hospital admission he had developed severe pain in the right foot at night while supine in bed, the last week of which period he had tried without success to

sleep sitting in a chair. He stated that hot foot soaks aggravated his pain.

Physical examination on admission revealed a prematurely aged man in great pain. The positive findings were restricted to the right leg. His blood pressure was 110/78. When placed in the supine position his right lower leg to the knee became pallid and anesthetic to pin-prick in three minutes. In the dependent position the right foot slowly developed a profound cyanotic rubor. The right femoral, popliteal and pedal pulses were all absent. Peripheral pulses on the left were all present.

He was given 400 mg. etamon (tetra-ethyl ammonium chloride) intravenously, and moderate (80/40) hypotension promptly developed with the precipitation of severe right lower leg and foot cramps. The following day a diagnostic right lumbar ganglionic block was done. This again resulted in transient hypotension with precipitation of severe right calf and foot cramps.

Realizing that anything we did might not improve the circulation to the part, it was believed that unless this objective was achieved, a low thigh amputation would be mandatory. On March 2, 1948, a right lumbar sympathectomy (L2 through L4) was performed. The patient had an uneventful convalescence. Immediately after operation his severe right calf and foot pain at rest in the supine position was largely relieved.

By March 15, 1948, he could sleep only in the supine position for two to four hours before the right foot would become numb, although no blanching was noted. He improved slowly over the next few weeks and by May, 1948, was able to walk slowly three blocks with some fatigue and discomfort. The tips of the toes of his right foot were 6 to 8 degrees warmer than the left. A peculiar phenomenon appeared at this time. This consisted of explosions of severe burning pain over the lateral aspect of the right foot and leg. The patient obviously was not rehabilitated for useful employment. Now an arteriectomy (as suggested by Leriche) was considered.

On June 30, 1948, resection of a completely obstructed right common iliac artery was performed. At this time all of the peripheral pulses on the left leg were present and of good quality. Immediately following this operative procedure the explosions of pain in the right lower leg and foot disappeared. His effort tolerance promptly improved and by August, 1948, he had returned to a full eight-hour shift as a machinist.

By January, 1950, he showed further improvement. He had no claudication walking at a moderate gait but would awaken twice nightly with a sense of band-like constriction around the first and second toes of his right foot. In March, 1950, he was hospitalized at Madigan General Hospital (Fort Lewis, Washington) with a mild coronary attack. He returned to work and was seen two months later with no complaints referable to a warm, dry foot that showed only minimal cyanotic rubor in dependency.

The next follow-up examination was in November, 1952. Six months before he had been admitted to the Veterans Hospital in San Francisco with a complaint of progressive intermittent claudication in the left calf and foot. A left lumbar sympathectomy was carried out at this hospital. He now had bilaterally absent femoral pulses but was working a full eight-hour shift without discomfort in his legs. The legs and feet were warm and dry. There was very little cyanotic rubor in dependency. He was experiencing some anginal pain on effort, and four months before had had a mild cerebrovascular accident with a short, transient period of speech aphasia.

On July 26, 1954, some six and a half years since first seen, he was admitted to the Veterans Hospital, Seattle, with ulceration of the tip of the left fourth toe, marked bilateral cyanotic rubor and leg edema. Death followed in a few weeks after an acute coronary thrombosis.

This case has been classified in the accompanying statistical study as a "fair to good" end result.

CASE II. A. F., a sixty-four year old woman, was first seen on February 7, 1950. Her chief complaint was pain in both calves and feet on exercise for the preceding three years. At the onset the pain of claudication was precipitated by walking two blocks. For the last two months walking about her small house was all the effort needed to bring on the pain. She had been unable to sleep supine for the two weeks prior to this first examination. Her left foot had a sense of numbness and both feet had become more pallid. She noted that warm foot soaks increased her distress.

Physical examination was entirely negative except for the lower extremities. The femoral

pulses were present, but the left was of much greater volume than the right. The pedal and popliteal pulses were bilaterally absent. There was marked blanching of the feet on elevation, slow venous filling in dependency and marked cyanotic rubor. Both ankles and feet were edematous from the preceding two weeks of constant leg dependency.

On February 10, 1950, bilateral lumbar sympathectomy (L2 through L4) was performed. The postoperative course was without event. Immediately the patient was able to sleep supine without pain. By March 6th she was able to walk one block without distress and all sense of numbness in the feet had disappeared. It was interesting to note at this time that digital skin temperature on the right averaged 94°F., while on the left the temperature was only 77°F. In April (three months postoperatively) the digital temperatures were equal bilaterally (90+°F). Intermittent claudication was precipitated by walking two blocks.

On October 30, 1950, she spent the entire day shopping, with no leg or foot distress.

On October 6, 1954 (approximately three and a half years postoperatively), she could still walk three blocks before having claudication and could resume walking after three minutes' rest. Her feet were warm, dry and pink. There was little or no blanching on elevation, or cyanotic rubor in dependency. The femoral pulses were bilaterally present. The toes and sole of the right foot averaged 94°F.; left, 89°F.

This case has been classified in the statistical study as an "excellent" end result.

Case III. W. G., a fifty year old white man, was first seen October 19, 1949. His chief complaint was pain in the left calf on effort with numbness and tingling of the left foot and toes of two weeks' duration.

He stated that for the past year he had from time to time been awakened at night by left foot cramps. There had been no associated history of intermittent claudication.

On September 1, 1949, he left on an automobile trip from Seattle to Mexico City. Enroute he reached Dodge City, Kansas, stepped out of his car to walk, and for the first time noted that after walking two blocks severe cramps developed in his left calf and he had to stop. By the time the trip was nearing its end, approximately two or three weeks prior to this examination, pain on walking in his left calf and foot became more severe and was present with little or no effort. The pain extended well up into the mesial left thigh. He noted his left foot was cold to touch, and was aware of numbness and tingling in the toes of his left foot.

Physical examination revealed no abnormalities except in the lower extremities. The right leg was normal on inspection. Both feet were cold on palpation. No popliteal or pedal pulses were felt in the left leg. The left femoral pulse was only approximately one-sixth the volume of the right femoral pulse. There was moderate blanching of the left foot on elevation. Cyanotic rubor was not noted, but the return of color to the pallid foot was markedly delayed. With the examining room at 70°F., his digital temperatures were bilaterally equal at 74°F. He was given 500 mg. etamon intravenously, with a 14°F. temperature rise on the right and no measureable elevation of skin temperature on the left.

On October 25, 1949, left lumbar (L2 through L4) sympathectomy was performed. His postoperative course was uneventful. On examination November 11, 1949, he was completely relieved of all distress on effort. The digital temperature of the left foot now averaged 92°F; right, 73°F.

On May, 1951, he experienced a mild cerebrovascular accident that promptly subsided without residue.

On August 24, 1954, he was re-examined. He stated then that if he walked briskly for one block he noticed some slight pain in his left hip which subsided promptly with a moment's rest. There was no further calf pain, numbness or tingling in the left foot or toes. Elevation of the left leg caused no foot blanching. He had no cyanotic rubor in dependency. The left femoral pulse was barely palpable and the left pedal pulses were absent.

This case has been classified in the accompanying statistical study as Arteriosclerotic Peripheral Arterial Insufficiency Associated with Relatively Recent Acute Arterial Thrombosis. It was further classified as an "excellent" end result.

CONCLUSION

Fifty-three cases of obliterative arterial disease of the lower extremity representing eighty-one lumbar sympathectomies have been appraised. It would seem that the evidence of

the worth of this procedure in properly selected patients is further strengthened.

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DISCUSSION OF PAPERS BY DRS. PAYNE AND WINSOR, MOVIUS AND WEINBERG, AND MACMAHON

Emile F. Holman (San Francisco, Calif.): We are indeed indebted to Drs. Payne and Winsor for the ingenious methods and devices to determine the extent of arterial insufficiency either from obliteration or from spasm. They will undoubtedly contribute greatly to the accurate appraisal of this obliteration and thereby serve to determine treatment more precisely. As to the number of ganglions to remove, one is confronted all too frequently with anatomic limitations. Personally, I want to dilate as many vessels to a limb as possible and have consistently removed the fourth to the second ganglions; that is when I have been able to recognize them! There is an extraordinary variation in the anatomic configuration of the lumbar sympathetic chain. Occasionally they are all amalgamated in one, sometimes in two, and only rarely can one differentiate one ganglion for each lumbar vertebra.

A question that is constantly before us is how long does the improvement last which follows sympathectomy. A few cases may suffice as examples of what we may expect. In 1939 sympathectomy was performed upon a thirty-eight year old woman with bilateral arterial obliteration due to a very localized atherosclerosis of the terminal aorta. She is well today, restricted in activity to be sure, but living a fairly normal life with no evidence of sympathetic reinnervation. In another woman, sixty-six years of age, bilateral lumbar sympathectomy was performed in 1948 for obliteration of the terminal aorta which prevented her walking more than 10 feet without painful claudication. She is well and doing her own shopping today, at the age of seventy-three.

To the authors, our thanks for this instructive paper.

John K. Poppe (Portland, Ore.): Dr. Movius is to be congratulated on his fine presentation with particular emphasis on the critical analysis and detailed description of his complications, which is a great deal more educational than merely emphasizing his successes.

Although I have been interested for a long time in reinforcing aneurysms by the application of irritating substances externally, my experience with resections has been primarily with lateral resections of saccular aneurysms by clamping and the insertion of plastic prostheses in the aorta. This is at least partly due to the inadequate supply of suitable homologous artery grafts in the private hospitals of Portland at the present time. Since this situation may apply elsewhere, perhaps the use of plastic grafts may represent the answer to this problem.

The presence of a collection of rigid plastic tubes of methyl methacrylate of assorted sizes, such as those introduced by Hufnagel, have proved extremely comforting when complications arise in resecting coarctation of the aorta. This particular type of graft occasionally has certain advantages over other types in which suture anastomoses are required. Perhaps some others have had the distressing experience of having the attenuated sclerotic wall of a dilated segment of aorta in an overaged patient practically disintegrate before their eyes as the fine silk sutures tear out and the tear continues to extend up into the sclerotic subclavian artery, which would represent the only other source of a bridge or graft. It is in this situation that the multiple point fixation ring employed with the Hufnagel tube has provided an opportunity for successful restoration of continuity of the aorta. These tubes are 2½ inches in length as manufactured by the Brunschwig Company,* which is very much the same as the plastic tube containing a ball valve used in the same location for aortic valvular insufficiency. The only patient in whom such a tube has been used by me has survived eight months to date.

* Source of methyl methacrylate grafts: Brunschwig Manufacturing Company, 61 Hampshire Street, Boston, Mass.

Saccular aortic aneurysms with a neck short enough to permit clamping seem relatively rare but respond well to lateral resection, as suggested by Bahnson. Due to difficulties in finding room for the handles of clamps long enough to cover the neck of such aneurysms, a special clamp with 16 cm. blades without handles was made for me by the Pilling Company. This clamp has multiple fine teeth to prevent slipping and has bolts on the ends for approximation of the blades. The application of two coarctation clamps from the sides leaves a considerable leak between the ends of the clamps. This requires a temporary interruption of blood flow through the aorta. When this occurs in the thoracic portion of the aorta, I have suspected that it may be responsible for some of the postoperative diarrheas of which these patients complain. One such patient was noted to have innumerable small superficial ulcerations of his rectal mucosa on postmortem examination following intractable diarrhea.

Dr. Movius' suggestion of injecting increased amounts of heparin in the distal segment of aorta or iliac arteries before clamping is well taken and may prove helpful. I intend to apply this to my experimental work on dogs with orlon fabric grafts which have shown a tendency to become occluded with retrograde thrombosis despite satisfactory function at the completion of the operation. I have used heparin sparingly in these dogs and inactivated that part left on the graft with protamine to avoid undue leakage through the porous cloth, but there would be no contraindication to the use of larger amounts in the distal segments of the vessel. Dr. Hufnagel's recently published remark that his orlon grafts no longer become obstructed by thrombi since he began to wash the fabric thoroughly with detergents before using it internally may suggest another answer to my problem of thrombosis.

I appreciate the opportunity to discuss this interesting and informative paper.

Frederick L. Reichert (San Francisco, Calif.): Drs. Payne and Winsor remove only the one ganglion and I think perhaps they are right; during the depression years patients could not even come into the clinic wards to undergo sympathectomies and I had to block them with alcohol. I tried one, two, three needles and then I tried only one, with 1 to 2 cc. of 95 per cent alcohol, and some of these sympathetic blocks lasted four to five years.

I have been treating Buerger's disease since 1920 and all of my patients perspired. When the lumbar sympathetics are removed, the torso perspires. Why? We think now that the disease is just as extensive as any other vascular disease, extending throughout the entire body, and the patients are sensitized. We have these patients dry now, with reserpine or Rauwolfia root which suppresses the sympathetic center in the hypothalamus.

Jack A. Cannon (Los Angeles, Calif.): Dr. Movius is to be complimented on his very enviable record in dealing with this difficult problem. I want to mention briefly our experience with this problem at the U.C.L.A. Medical Center. Among the fifteen patients treated by four different surgeons, our results have been as follows:

Nine patients are alive and well. One of these was operated upon by Dr. Harry Muller nearly fourteen months ago for acute rupture with massive exsanguination. I recently operated upon another patient in this group for acute rupture of the aneurysm and so far his condition is good.

However, there were six deaths. Three occurred in the immediate operative period: One of these patients, who was operated upon in deep shock for rupture of his aneurysm, was resuscitated from two cardiac arrests during surgery but did not survive the slow and very gradual removal of the proximal occluding clamp once the anastomosis was completed; one death resulted from leakage of the proximal suture line a few hours after operation; the other death followed hemorrhagic shock secondary to surgical trauma to the vena cava, which was ultimately controlled but not until irreversible changes had occurred. Three late postoperative deaths were encountered: In one the homograft itself ruptured two and a half months after operation; in one leakage from the proximal suture line occurred three weeks after operation and was recognized and corrected, but the patient died from his pre-existing renal disease; the third patient was operated upon in deep shock for a ruptured aneurysm and survived ten days but died of unrecognized gangrene of a segment of his sigmoid colon.

In five of the total fifteen, nylon prostheses were used. Only one of these patients is alive and well. Another is alive four and a half months after operation but has a recurrent false aneurysm which we believe is the result of leakage from the proximal suture line. He is asymptomatic but the aneurysm appears to be growing. One patient died of late leakage from the proximal suture line. One patient died following the two cardiac arrests already mentioned, and one died of immediate leakage from the suture line.

It is my belief that the indications for operation in this condition are in the phase of definition. I do not believe that the mere discovery alone of an abdominal aneurysm is an indication for surgery because most of these patients are unfavorable operative risks and the mortality of operation is certain to be high. At present I would recognize two main indications: (1) evidence of impending rupture in the form of enlargement or onset of

pain; (2) actual rupture. Once diagnosed, rupture appears to be an absolute indication since otherwise death is practically certain.

HERBERT J. MOVIUS, II (closing): Dr. Holman stated that in their hands longitudinal incision to increase circumference had been a satisfactory procedure. I realize that to draw conclusions from a single case is dangerous, and this is what I have done. However, in the one graft in which this was used, disruption of the suture line occurred at the apex of the incision and has made me believe this procedure should be avoided if possible. Dr. Cannon stated that they were not yet certain which patients are candidates for aneurysm resection. We possibly are a little less conservative in choosing patients for resection of their aneurysm, but we certainly do not believe that the presence of an aneurysm alone is indication for surgery.

Conversion of Billroth II to Billroth I for the Relief of Postgastrectomy Symptoms

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A GREAT many surgeons through the years have advocated gastroduodenostomy rather than gastrojejunostomy as a means of restoring gastrointestinal continuity following gastric resection, believing it to be a more physiologic procedure and one less likely to lead to serious postgastrectomy symptoms. Only a few, however, have considered the conversion of gastrojejunostomy to gastroduodenostomy for the relief of these disabling symptoms.[1,2,3,6,7,9,11,15] Because it is generally agreed that the greater the amount of stomach removed when treating peptic ulcer the less likelihood there will be of recurrent or marginal ulceration, most surgeons believe that gastroduodenostomy usually cannot be accomplished safely after an adequate amount of stomach has been removed. On the other hand, it is apparent that the greater the amount of stomach resected, the greater the possibility of serious postgastrectomy difficulties.

Unfortunately, there is little agreement about the frequency and type of symptoms which follow subtotal gastrectomy. The incidence varies in each published series, depending upon the criteria adopted for the selection of cases and upon the keenness of the investigator.[10] There can be little doubt, however, that digestive and other symptoms do occur and, at times, constitute a serious problem in postoperative management.

Actually, there is a wide variety of symptoms which occur following gastric surgery.[10,13] Some are extremely mild and fleeting while others are sufficiently distressing to cause partial to total disability. Occasionally, but fortunately rarely, death ensues. In an analysis of symptoms following subtotal gastrectomy Muir[10] found them to fall into the following groups in order of incidence and importance: (1) the "dumping syndrome"; (2) failure to gain weight; (3) psychoneurotic symptoms; (4) bilious regurgitation and vomiting; (5) hypoglycemic attacks; (6) deficiency syndromes. In view of this variety of complaints, it is not surprising that different causes have been ascribed to the various patterns of symptoms encountered.

Although it is not within the scope of this paper to review the many and varied opinions as to the cause of these various states, it is important to emphasize certain anatomic and physiologic considerations that may have some bearing upon the subject under discussion.

Two general methods are available for the re-establishment of gastrointestinal continuity after resection. These methods, as pointed out by Moore and Harkins,[8] involve utilization of either a gastroduodenal anastomosis (Billroth I and its modifications) or a gastrojejunal anastomosis (Billroth II and its modifications). The Billroth I type of gastrectomy leaves the upper gastrointestinal tract in an anatomic situation more closely simulating the normal. The gastric contents, rather than being directed into the jejunum, take the normal course through the duodenum, thus promoting the normal physiologic stimulation of pancreatic, biliary and hepatic functions. Emptying of the stomach into the duodenum usually takes place more slowly than into the jejunum, permitting a more normal period of digestion in the proximal loops of intestine. Experimental evidence also indicates that the presence of acid in the duodenum exerts an inhibiting effect upon the secretion of acid by the stomach, thus protecting the duodenum against stomal ulceration.[12]

In all modifications of the Billroth II operation ingested food is directed into the jejunum and therefore does not pass through the duodenum. As a consequence, the normal physiologic responses to the stimulation of gastric contents upon duodenal mucosa do not take place. Essential aids to normal digestion are

impaired. It is believed that the mobility of the duodenum is controlled, at least partially, by the passage of gastric contents into it.[3] Without this stimulus duodenal ileus is apt to develop, a factor thought by many investigators to be of paramount importance as a cause of the dumping syndrome.[8] The rate of flow and degree of emptying of the gallbladder and the secretion of pancreatic juice are regulated not only by the presence of acids, fats and bile in the duodenum, but also by the stimulus initiated by the contact of food with the duodenal mucosa. There seems also to be a considerable degree of correlation between the rate of inflow of bile into the duodenum and the rate of flow of pancreatic juice. It is evident, therefore, that the lack of normal physiologic stimulus of gastric contents upon the duodenum may lead to impaired emptying of the gallbladder, together with inadequate hepatic, duodenal and pancreatic secretions. Furthermore, the rapid passage of food through the small gastric remnant and proximal bowel into the lower reaches of the small intestine does not allow adequate contact with the normal secretions of the upper small intestine for normal digestion to take place. It is not surprising, therefore, that the resultant failure of the normal physiology for digestion often leads to diarrhea, steatorrhea and failure to gain weight, or in severe cases to a continuous loss of weight. Furthermore, the iron deficiency anemias which result frequently from gastric resections may be explained partially on the lack of absorption of iron by the duodenal mucosa.[1,6] Fortunately, in most instances these physiologic deficiencies are compensated adequately and the patient remains relatively well; however, at times compensation is inadequate and those patients endure a miserable existence regardless of all medical aid.

The relationship of postgastrectomy symptoms to the type of gastrectomy performed possibly has not received the consideration it deserves. There can be no doubt that symptoms occur following all types of gastric surgery, the severity and duration depending upon a great many variable factors. In recent years the emphasis by most surgeons has been directed largely toward the cure of the ulcer and the prevention of stomal ulcers. Even though they agree that postgastrectomy symptomatology is less severe and of shorter duration following gastroduodenostomy, its application, except in the hands of a few, has been limited to those patients with gastric ulcer and to those with proximal duodenal ulcer without fixation and with a mobile duodenum.[13] There being no limit to the amount of stomach that can be resected and continuity established safely by gastrojejunostomy, this has become the procedure of choice.

A number of modifications of the original Billroth I type of gastroduodenal anastomosis have been advocated with the idea that its usefulness might be more widespread.[6,7,9] Most of these involve methods devised to mobilize either the stomach or the duodenum more adequately or to alter the type of anastomosis.[8,13] More recently a less extensive gastrectomy, gastroduodenostomy and vagotomy has been advised.[5]

Others have recommended the interposition of a segment of large or small bowel between the cut ends of the stomach and duodenum, particularly when for anatomic reasons direct anastomosis cannot be accomplished without tension.[6,7,9]

All of the modifications have been advocated mainly to accomplish gastroduodenal anastomosis at the time of original gastric resection rather than as a conversion procedure once gastrojejunal anastomosis has been accomplished and has failed.

In 1935 Perman, in a discussion of gastritis, intestinal trouble, asthenia and anemia after operation for ulcer, described disabling symptomatology following the Billroth II type of resection in three patients, all of whom were reoperated upon and had conversion to the Billroth I type, with almost complete relief of symptoms in two and death from gastric ileus in the third. The first patient was reoperated upon in 1929. Again in 1947 Perman[11] recommended that "in severe cases of dumping syndrome after resection the best treatment is to change them to a stomach of the Billroth I type." He has performed this operation in twenty-five cases since 1929 with one death and claims that a large number of the patients were cured completely following operation. In only one patient was there a recurrence of duodenal ulcer.

In 1950 Bohmansson,[1] in a discussion of the prophylaxis and therapy in late postgastrectomy complications, reported thirty-six cases in which good results were obtained with reoperation consisting of restoration of the physio-

logic duodenal passage. He claims to have reported good results as early as 1927.

In May, 1951, Moroney[9] published a preliminary account of twenty-two patients (twenty with peptic ulcer and two with postgastrectomy symptoms) submitted to operations in which a segment of transverse colon was interposed between the gastric segment and the duodenum. All of the patients made satisfactory recoveries and were reported as well.

In 1952 Henley[6] published his preliminary report of a fundamental change in the technic of gastrectomy which would "restore the continuity of the alimentary tract and be applicable to all lesions of the duodenum, stomach and esophagus and to those cases of severe so-called postgastrectomy syndrome consequent upon the Billroth II operation." He transplants a segment of jejunum in a retrocolic position between the stomach and duodenum. He reports jejunal replacement of part or of the whole of the stomach in thirty-five patients without mortality. Four patients were operated upon for and relieved of postgastrectomy symptoms. None, however, had been followed up for an appreciable length of time.

Clagett and Waugh[3] in 1948 contended that fixation, shortening and narrowing of the duodenum usually would not permit a safe and satisfactory restoration of gastroduodenal continuity after gastric resection. However, they recommended its use in cases of gastrojejunal ulcer when the original duodenal ulcer had healed and the duodenum again appeared normal.

In 1951 Teicher, Friedman and Lipton[15] reported the case of a fifty-two year old man who had suffered from severe vomiting and loss of weight following an antecolic, antiperistaltic Polya type of subtotal gastrectomy. Eight months later, because of continued weight loss, vomiting and diarrhea, the patient was reoperated upon and an additional amount of stomach removed. This time an antecolic, isoperistaltic Polya type of anastomosis was performed. Two weeks later the patient's symptoms recurred and persisted. After continued pain, vomiting and loss of weight for an additional eight months, the Billroth II procedure was converted to a Billroth I, with complete relief of symptoms. Eight months later the patient was maintaining a weight gain of 40 pounds and was no longer vomiting.

It is certain that many other surgeons have had this same experience in isolated cases which they have not reported.

Dr. Lyle Hay, in a discussion of a number of papers concerned with gastric surgery at the meeting of the Society of University Surgeons in February, 1952, stated that in two cases he had converted a Billroth II to a Billroth I type of anastomosis for severe dumping syndrome. His follow-ups suggested that both patients were going to be relieved of symptoms.

Bell[2] has mentioned one patient whose symptoms were so severe that this procedure was undertaken.

Our own experience has been extremely limited but so dramatic in two cases that it seems worth while to report them.

CASE REPORTS

CASE 1. This patient was a sixty-six year old, Italian born, retired cement worker who first developed stomach trouble four years before his entry to the San Francisco City and County Hospital on February 16, 1953. In February, 1951, he had been operated upon at another hospital where a retrocolic antiperistaltic Polya modification of the Billroth II type of gastric resection was performed for a bleeding duodenal ulcer. Soon thereafter he was reoperated upon for an evisceration. There was some relief of abdominal pain for about four months; however, he failed to gain weight and strength, and had no appetite. He was unable to return to work. In July, 1951, epigastric distress and nausea after meals again developed. He noted a loss of 25 pounds in weight in the few months since operation. In August, 1951, he entered the San Francisco City and County Hospital for the first time, complaining of epigastric distress and jaundice. A diagnosis of homologous serum jaundice was made. He was discharged after five weeks of hospitalization, clear of jaundice but unable to gain weight. A gastrointestinal series in September, 1951, revealed evidence of a partial gastrectomy with approximately two-thirds of the stomach remaining. A satisfactorily functioning gastrojejunostomy without evidence of stomal ulcer was demonstrated. He was followed up in the clinic, where it was noted that he continued to complain of epigastric pain. In January, 1952, a gastrointestinal series again revealed no evidence of marginal ulcer. In November, 1952,

he was admitted again to the San Francisco City and County Hospital after having passed gross blood from the rectum for three days. Hemoglobin was 4 gm. per 100 cc. He had again lost 25 pounds in weight and x-ray examination revealed a probable marginal ulcer. He was discharged after treatment with diet, iron and blood transfusions. In February, 1953, he again entered the hospital, stating that he had had another bright red rectal hemorrhage five days after leaving the hospital the previous time. He had remained on a diet and under the care of a private physician but continued to have nausea, dyspnea and epigastric pain. A gastrointestinal series revealed a functioning gastroenterostomy with a stomal ulcer. On March 11, 1953, he underwent another operation. A marginal ulcer was demonstrated and resected. Two-thirds of the remaining stomach were resected and a modified Billroth I type of gastroduodenostomy accomplished after an end-to-end closure of the resected portion of the jejunum had been undertaken. In the first six months following this procedure the patient complained of fulness and distress following meals. His appetite and general well-being improved gradually and he regained his normal weight. In November, 1954, twenty-one months after the conversion procedure, he re-entered the hospital for excision of a hydrocele. An upper gastrointestinal series revealed a Billroth I type of gastroduodenostomy which functioned normally. No evidence of gastric or stomal ulcer was demonstrated. In the five-hour film the head of the barium column was still in the small bowel. There was no barium residue in the stomach. The patient now was free of gastric symptoms and back to his normal weight.

Comment. This case demonstrates a bleeding duodenal ulcer, not relieved by two years of medical treatment, that was subjected to an inadequate gastric resection of the Billroth II type. Soon after operation symptoms of recurrent ulcer developed which subsequently caused severe gastrointestinal hemorrhage. Following resection of the marginal ulcer and an adequate gastric resection, gastrointestinal continuity was re-established by gastroduodenal anastomosis. Postprandial epigastric distress occurred in the immediate postoperative period, requiring frequent small feedings, but the patient gained weight and strength, was relieved of pain and after six months became asymptomatic. When last seen twenty-two months after the last operation he was well.

CASE II. This patient was a forty-four year old unemployed man who entered the San Francisco City and County Hospital in November, 1953, complaining of constant epigastric pain, persistent vomiting and loss of weight after an operation for similar symptoms in October, 1953. The past history revealed an old tuberculous arthritis of the right knee for which he had had numerous operations. An appendectomy had been performed in childhood. The patient first started having stomach trouble in May, 1952, and was operated upon in July, 1952, at the Doctors Hospital in San Francisco. A report from his surgeon states that a Mayo-Polya isoperistaltic antecolic modification of the Billroth II type of subtotal gastric resection was performed for a benign gastric ulcer. The operation resulted in partial relief of symptoms for about six weeks. However, epigastric pain and vomiting then recurred and persisted until July, 1953, when a cholecystectomy for cholelithiasis was performed by the same surgeon. Thereafter the symptoms of epigastric pain and vomiting persisted. In August, 1953, he reported to the Riverside Community Hospital in southern California. A note from his physician reports that there was some symptomatic relief from diet therapy, antispasmodics, phenobarbital and antacids for a period of five weeks. However, the pain and vomiting soon returned and were relieved only by opiates. A gastrointestinal series at that time revealed a questionable filling defect in the remaining stomach. In September, 1953 he underwent operation. There was no evidence of gastric malignancy but there was some induration of the gastrojejunostomy stoma, suggesting a marginal ulcer. Bilateral vagotomy was performed. There was recurrent vomiting in the second week after operation. The patient did not respond to dry diet, urecholine, liquid diet, etc. He was discharged, only to return with persistent vomiting, weight loss and weakness.

In October, 1953, a gastrointestinal series revealed gastric retention with nearly complete obstruction at the gastroenterostomy. No marginal ulcer was demonstrated. Gastric analysis revealed 26 units total acid, negative free acid.

In October, 1953, he again underwent opera-

tion. The entire stomach and small bowel were freed of adhesions. The proximal jejunal loop was found to be approximately 24 inches in length, there having been an antecolic, isoperistaltic Polya type of anastomosis. A segment of jejunum was resected, making the proximal loop not over 6 inches in length. Again the postoperative course was marked by vomiting and epigastric pain which persisted until his entry to the San Francisco City and County Hospital in November, 1953. Upon entry the patient was complaining of constant severe epigastric pain and vomiting. He had lost weight from a normal of 150 pounds to 70 pounds. He was severely malnourished and was suffering with nutritional edema. He was unable to retain liquids or food taken by mouth. A gastrointestinal series revealed subtotal gastric resection of the Billroth II type with a normally functioning gastrojejunostomy and no evidence of stomal ulcer. At five hours the head of the column of barium had reached the terminal ileum, with the remainder scattered throughout the small bowel.

Neuropsychiatric survey revealed mental deficiency but no psychiatric or severe neurotic problem. Gastroscopic examination was essentially negative. Because the patient showed little improvement on all types of dietary, medical and intravenous therapy, it was thought that he must have an undemonstrable active marginal ulcer. Repeat x-ray examination of the upper gastrointestinal tract again revealed a normally functioning Billroth II type of gastrojejunostomy, with no evidence of stomal ulcer and an empty stomach after three hours. Operation was performed on January 7, 1954. A high gastric resection with an isoperistaltic, antecolic, full lumen Mayo-Polya modification of the Billroth II type was found. The proximal jejunal loop measured 8 cm. and the old end-to-end anastomosis accomplished at the third operation was patent. The gastric remnant was estimated to represent about 20 per cent of the original size of the stomach before resection. In the absence of demonstrable disease it was decided to convert the Billroth II anastomosis to a Billroth I, utilizing a segment of jejunum for interposition between the gastric and duodenal stumps. This was accomplished readily by transecting the jejunum at its juncture with the greater curvature side of the gastrojejunostomy and closing the proximal end. The proximal jejunum then was transected at the point of the old end-to-end anastomosis near the ligament of Treitz. The proximal and distal segments were anastomosed end-to-end. The remaining open end of the jejunum attached to the stomach was anastomosed to the duodenal stump, which had been mobilized and reopened. This left an antecolic segment of isoperistaltic jejunum about 6 cm. in length interposed between the stomach and duodenum. The postoperative course was uneventful. On the nineteenth postoperative day a gastrointestinal series showed some delay of emptying of the gastric remnant into the jejunal segment, but none from the jejunal segment into the duodenum. At four hours there was still some gastric residue. The patient made a most remarkable recovery. All vomiting and pain disappeared after surgery. He began eating and gaining weight almost immediately, and was discharged on the twenty-second postoperative day feeling well. One month after his discharge he was eating three meals a day and had no complaints. His weight had risen from 70 to 116 pounds. A gastrointestinal series in March, 1954, approximately three months postoperatively, showed essentially the same picture as that three weeks postoperatively, with delayed emptying of the stomach. By October, 1954, however, nine months after the conversion procedure, he had no postgastrectomy symptoms and had gained weight to 141 pounds. A gastrointestinal series in December, 1954, showed some reflux of barium from the stomach into the esophagus and delayed emptying of the stomach. At four hours there was approximately 10 per cent gastric residue with distribution of the remaining barium throughout the small bowel. Although the patient had had an episode of diarrhea which lasted about two weeks, causing a 10 pound loss in weight, he had recovered and was again in good health and working.

Comment. This case represents that type of postgastrectomy symptomatology manifested mainly by epigastric pain, vomiting and severe loss of weight. These symptoms developed shortly after gastrectomy of the Billroth II type and were not relieved by various types of medical treatment, cholecystectomy, revision of the Billroth II type of gastric resection with shortening of the afferent jejunal loop, or by vagotomy. Conversion of the gastrojejunostomy to a gastroduodenostomy with interposition of a segment of jejunum resulted in imme-

diate relief of pain and vomiting, and initiated a prompt and pronounced gain in weight.

CONCLUSION

Neither of these two patients has been watched for a sufficient period of time not to fear the possible development of recurrent duodenal or jejunal ulcer. This would seem to be the most likely late complication that might reasonably be expected. Nevertheless, the dramatic relief of symptoms together with the rapid gain in weight and strength, particularly in the second patient, makes this procedure one well worth consideration for the relief of serious postgastrectomy symptomatology.

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DISCUSSION

Leon Goldman (San Francisco, Calif.): Dr. Mathewson has reported on two patients with "postgastrectomy symptoms" following a Billroth II operation whose symptoms were relieved following conversion to a Billroth I.

In reviewing the details of the first case, namely, that a bleeding jejunal ulcer occurred following a Billroth II type resection of the stomach, one is impressed with the fact that only approximately one-third of the distal stomach was removed at the original operation. The most important part of the procedure carried out by Dr. Mathewson on this patient was the completion of an adequate gastric resection. This may have relieved the patient of all untoward symptoms, but certainly it has been proved that gastroduodenostomy affords a better nutritional state postoperatively.

The second patient experienced epigastric pain and vomiting following a Billroth II procedure with adequate gastric resection. Subsequent vagotomy and lysis of adhesions proved to be ineffective. Our experience dictates that a new stoma must always be constructed to obtain relief of such obstructive symptoms. This patient did very well following conversion to a Billroth I procedure. The symptoms which had been present ever since the initial operation were those suggesting a malfunctioning stoma or some degree of physiologic or mechanical obstruction about the stoma or the jejunal loops. In such cases it is rather commonplace to be unable to locate an anatomic obstruction at operation. In this case it is likely that an obstruction was corrected during the conversion procedure.

In both of these patients postgastrectomy symptoms—a jejunal ulcer in one and some mechanical or functional obstructive phenomenon in the other—resulted in marked malnutrition, vomiting and epigastric pain, which were readily corrected by the conversion procedure.

Since the different types of gastric resection for the treatment of peptic ulcer have been more critically evaluated, surgeons are free to admit the occurrence of untoward symptoms following gastric resection. Dr. Mathewson emphasizes that the so-called dumping syndrome is only one of many types of symptoms; it did not exist in either of these patients. Without going into a discussion of the respective merits of the Billroth I and Billroth II types of operation, it may be said that the true dumping syndrome seems to be related to the amount of stomach removed, although its exact cause is not clear. The symptoms are apt to be more marked and of a higher incidence in patients with a wide resection than in those with a more limited one. Wangensteen reports an incidence of 20 per cent following the Billroth II type of resection. Henry Harkins and associates, in a critical analysis

of 196 of their own cases, reported that 34 per cent had some degree of dumping symptoms following a Billroth I procedure. These symptoms are characterized by postprandial sweating, flushing, nausea and palpitation relieved by lying down. In two-thirds of this group symptoms persisted after one year. This presents another aspect of the postgastrectomy syndrome; and if there is a substantial incidence as they report, it brings up the question of whether conversion would alleviate these symptoms. In 1950 Bohmansson reported excellent results following this procedure in nineteen of twenty-four patients afflicted with dumping symptoms following a Billroth II type of operation. Further experience and reports will tell us the value of the procedure for this syndrome.

DAVID METHENY (Seattle, Wash.): I prefer the Billroth I operation, but the interposition of small bowel between a gastric remnant and duodenum may have a discordant note that I want to bring before you.

Dr. Merendino has been placing portions of small bowel (in dogs) between gastric remnants and the esophagus. Ulcers have not developed even when stimulated with histamine. Putting the intestine between the stomach and duodenum is another matter. Dr. Henley, on his recent visit to Seattle, told us that in about 10 per cent of the patients upon whom he had performed this latter procedure ulcers had developed within a year. If I remember, his series had increased to about fifty. This is hardly surprising as the Roux-en-Y procedure, especially if followed by histamine stimulation, is one laboratory method of producing ulcers. In fact, any procedure produces ulcers which leads gastric secretions into other portions of the intestinal tract that are not neutralized by alkaline secretions.

Of course, Dr. Mathewson's two cases were emergency procedures, and the second patient had had a vagotomy. How this will alter the picture no one can yet say. In 1908 Sir Moynihan did a gastroenterostomy on a girl for an obstructing ulcer. In 1943, thirty-five years later, I happened to see her. She had been cured by that simple procedure until something else had come into the picture. Her relatives and friends in London were being bombed, and she was having symptoms of gastrojejunal ulcer for the first time. She was far too nervous, she said, to let me do anything about it; and when the bombs stopped, she apparently got better.

Surgery should overcome the disease for which the patient is being operated. It should also protect the patient from the stress that produced the original disease. Ideally, it should also prevent harm from the stresses which are yet to come. Conversion of a Billroth II to a Billroth I may be desirable, but insertion of small intestine between the gastric remnant and the duodenum seems to me to be an operation of necessity and not one of choice.

LESTER CHAUNCEY (Portland, Ore.): I would like to thank Dr. Mathewson for his fine paper. As always, it is excellent and we have learned something.

In our own practice we have converted anastomoses in two such patients. One taught us quite a bit. This was a woman who had had a pantaloon type of anastomosis, a type which is endemic in our part of the country. Subsequently, a marked dumping syndrome developed. She lost weight, going from 130 to 80 pounds. We converted her Billroth II anastomosis to a Billroth I as Dr. Mathewson has suggested. Following this she continued to have the same symptoms as she had had before; there was no improvement at all. She complained of abdominal pain and for this pain was taking so much tincture of opium and paregoric that her elimination became quite a problem, occurring about once every two or three weeks. Finally she was readmitted to the hospital. She was fed through an indwelling tube in her esophagus and, in addition, through an indwelling catheter which had been passed into the common iliac vein through the femoral. These measures helped a little, but actually she obtained the most help from a psychiatrist, who convinced her that her gastrointestinal tract could carry food, and from her husband, who threatened to divorce her. When he did so, she decided to stop all this nonsense; she began to eat quite well, gained weight and is well today.

We became so impressed by how helpful a good psychiatrist actually could be, that the second case we converted was done the other way around; we sent the patient to the psychiatrist first, before any operative procedure was performed. He gained the impression very quickly that her trouble was basically gastrointestinal and not emotional. Her Billroth II type of anastomosis was then converted to a Billroth I type and she did beautifully. I cannot stress the importance of the emotional side of this problem too much. Actually, I believe that most of the postoperative dumping syndromes that I personally have created have been in patients who in the light of present day thinking should not have been operated upon in the first place.

ALLEN M. BOYDEN (Portland, Ore.): I thought that you might be interested in a patient in whom I converted a Billroth I operation to a Billroth II. This patient was a sea captain who had been operated upon three times previously; first resection of an ulcer and pyloroplasty, then posterior gastroenterostomy and following this a Billroth I operation. He continued to remain a digestive cripple for some thirteen years. I saw him five years ago and, being impressed with our own results with the Billroth II type, performed the Hofmeister

modification of the Polya type of resection. He, too, as in the first case of Dr. Mathewson, had a stomal ulcer, having had symptoms ever since the Billroth I had been performed. He has remained perfectly well for five years. If he gets in trouble again, I may send him to Dr. Mathewson for another Billroth I operation.

CARLETON MATHEWSON, JR. (closing): I would like to thank the discussers. Although it is true that the Billroth I operation is regaining some of its former popularity, the so-called dumping syndrome occurs after the Billroth I procedure just as it does after the Billroth II procedure. Therefore I do not advocate the Billroth I in order to relieve the dumping syndrome. The really distressing symptoms which concern us most are not those of dumping, but are the persistent regurgitation and vomiting, loss of weight or inability to gain weight. These are matters of nutrition and may have something to do with the physiology of digestion which is disturbed when the stomach contents are diverted into the jejunum rather than passing through the duodenum.

I am interested in Dr. Metheny's comments regarding Dr. Henley's experience with jejunal ulcer. It would seem apparent that if one interposes jejunum between the stomach and duodenum, the chances of development of a jejunal ulcer would be great. Unlike a gastrojejunostomy where the protection of the alkaline contents of the duodenum bathing the anastomosis is present, a segment of jejunum between the stomach and duodenum is bathed in gastric contents without the alkalinizing factor. We have advocated this procedure only when it is not possible to reanastomose the stomach directly to the duodenum. Under these circumstances it is likely that the original gastric resection has been of such proportion that little acid secretion remains.

Dr. Chauncey's remarks are pertinent, but I can rise only to condemn the surgeon who sends his patients to psychiatrists for the treatment of postgastrectomy symptoms. Our second patient had been subjected to this type of treatment until almost dead. Reoperation with conversion to a Billroth I seems to have saved his life.

I believe that the failure of the Billroth I type of procedure in most hands may be attributed to the limited gastric resection that is undertaken in order to assure reanastomosis to the duodenum without tension. Under these circumstances, recurrence of the duodenal ulcer can be expected and can be relieved as Dr. Boyden has indicated, by an adequate resection and a Billroth II anastomosis.

Inguinal Hernia Complicated by Undescended Testis

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APPROXIMATELY 7 per cent, or one in fifteen, of hernias repaired at the Los Angeles Childrens Hospital in the last five years was accompanied by an undescended testis. With the present trend toward earlier surgery in both conditions, it seems worth while to review the experience of this hospital with the combined condition and to evaluate the evidence in support of the present trend.

When an uncomplicated hernia appears in a healthy newborn, or at any time later during childhood, surgical repair is now[8,10,13] indicated without delay, if hospital facilities and experience with the delicate structures of the inguinal region are available. The undesirable features of a truss are thus avoided and the recurrence rate is almost nil.

The optimal time for surgery in undescended testes, *per se*, has generally been considered to be at the age of eight to ten years.[1,4,10,18] Many have believed that as long as the condition is rectified by the time of puberty, no harm can be done.

Thus it has been the practice of the profession generally, and of this hospital, to defer surgery in the infant and young child with a hernia complicated by an undescended testis unless the hernia caused symptoms. This somewhat conservative attitude has been prompted primarily by the fear that in these tiny children the delicate structures of the cord[12] may be irrevocably damaged. It has been hoped also that in some of these cases partial spontaneous descent would occur and thus make orchiopexy an easier and safer procedure at a later time.

TECHNIC

A transverse incision is made (Fig. 1, inset) in the flexion crease and the skin and subcutaneous tissues incised down to the aponeurosis of the external oblique. This is opened in the direction of its fibers from the external ring upward. (Fig. 1.) The sac is exposed (Fig. 2), transected near the neck, opened and teased away (A) from the cord distal to the testis. Before its ligation it is dissected away from the vessels of the cord and vas (B), as these structures enter their peritoneal covered position. Gentle traction is placed on the testicle and cord (Fig. 3) by a traction suture placed in the gubernaculum. The fascial fibers which kink and shorten the cord are severed. A finger is pushed beneath Scarpa's fascia through the neck of the scrotum and the scrotal bed developed. (Fig. 4.) A small incision is made at the lower pole of the scrotum and a curved hemostat passed upward and attached to the gubernaculum. (Fig. 5.) By traction on the hemostat the testicle is then pulled to the very bottom of the scrotum. The silk suture which has been placed through the gubernaculum at its junction with the testis is attached to a rubber band fastened to the thigh.[3] (Fig. 6.) An alternate method (Fig. 7), the Torek[17] procedure, attaches the testis directly to the fascia of the thigh. This is separated three months later. When lengthening of the cord becomes difficult, section of the transverse and internal oblique muscles through the same incision at the internal ring (Fig. 8)[18] allows finger dissection of the peritoneum away from the intraperitoneal portion of the cord and adequate lengthening. This can be done early, between the ages of two and six, just as successfully as in the prepubertal child.

The testicle is placed in the scrotum, without fixation, only when it stays there without any hesitation (fourteen cases). The rubber band technic is used whenever there is the slightest retraction (fifty-seven cases). The Torek procedure is performed if this tendency to retract is more pronounced (fifty-five cases). The testicle

is fixed at the external ring and followed in a year or two with a second stage if there is real tension (five cases).

STATISTICS

Although almost all undescended testes are accompanied by a potential hernia,[18] prolapse of abdominal contents (clinical hernia)[19] into the vaginal process occurred 134 times in 201 cases of undescended testes[16] (an incidence of 66 per cent in this series). By the time the child reaches adulthood, Rusche[15] found the incidence of hernia to be 82 per cent.

Among 1,987 patients with clinical hernia operated upon at the Los Angeles Childrens Hospital between 1949 and 1954, there were 134 cases complicated by undescended testis. This gives an incidence of undescended testis in clinically manifest hernia in 6.7 per cent of the cases.

Age at operation was as follows: one month to two years in ten cases; two to six years, thirty-eight cases; six to eleven years, forty-eight cases; eleven to fourteen years, thirty-eight cases.

For special analysis the forty-eight patients operated upon at five years of age or less have been compared with the eighty-six older patients. Over half the younger group had a history of incarceration of the hernia, and

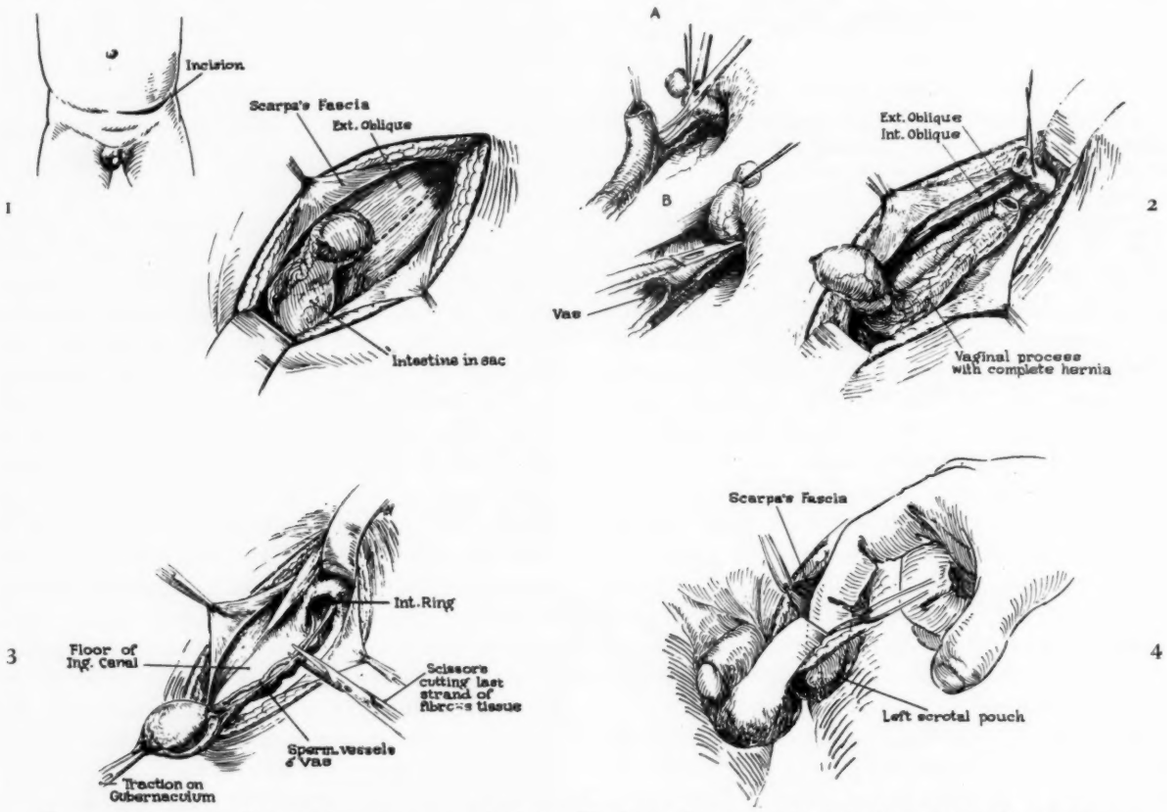

FIG. 1. The inset indicates a long transverse incision. The hernial sac containing testis and intestine is shown protruding through the external ring. Scarpa's fascia is lifted up, the external oblique exposed and the line of incision indicated.

FIG. 2. The main diagram indicates the opened external oblique with a complete hernia within the inguinal canal. The processus vaginalis has been transected and the upper end lifted upward. In A the distal portion of the vaginal process is being dissected away from the cord. In B the hernial sac is being freed from the cord, preparatory to ligation.

FIG. 3. The canal is again exposed with the sac ligated and the vaginal process removed. Fibrous bands within the cord structure are being severed to increase the length. Traction is placed on the gubernaculum to facilitate this.

FIG. 4. Scarpa's fascia is lifted up. A gloved finger is inserted into the rudimentary scrotal pouch, beneath the fascia and the left scrotal sac developed.

fourteen were actually incarcerated at the time of surgery. In three cases the testis was bluish or discolored[13] but was returned to the scrotum. None of these showed atrophy and in only one was there moderate retraction. In one case the hernia alone was repaired, with later surgery for the undescended testis. In all other cases both conditions were repaired simultaneously. The type of hernia repair was, in the main, simple ligation of the sac. In none of the cases of incarceration was it necessary to resect bowel. After lengthening the cord the testicle was simply placed in the scrotum in eleven cases, attached to the thigh with a rubber band in twenty-one and directly attached to the fascia of the thigh by suture[17] in sixteen.

Comments: All cases were followed up to five years. Forty-two have been checked from three months to five years. In one patient atrophy was present and in one, retraction. Thus in this group of operations performed early in life the anatomic results up to this time have been considered good in 96 per cent of the cases. There were no recurrent hernias.

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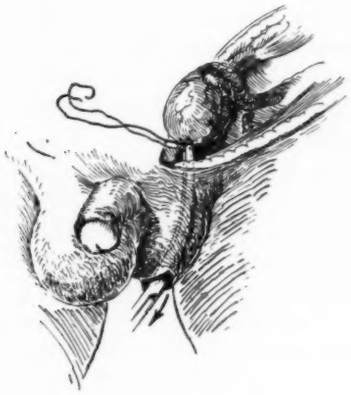

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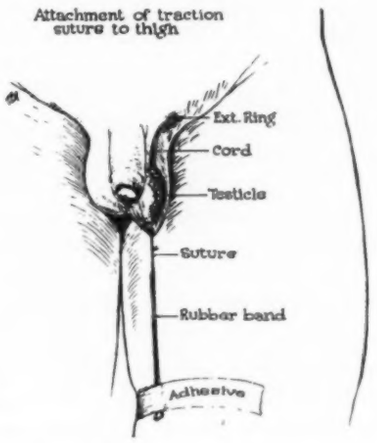

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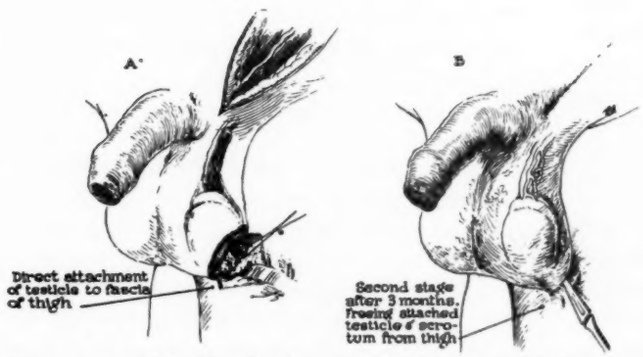
A
B
Direct attachment of testicle to fascia of thigh
Second stage after 3 months, freeing attached testicle of scrotum from thigh

8

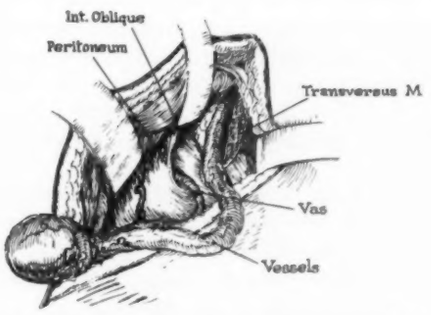

FIG. 5. A small incision has been made at the base of the scrotum, a curved hemostat inserted to grasp the gubernaculum with the attached suture. Thus the testicle is brought to the bottom of the scrotum.

FIG. 6. The opening in the scrotum has been closed. The suture which had passed through the gubernaculum is attached to a rubber band fastened to the thigh.[3]

FIG. 7. When there is slight tension on the testis after the cord has been lengthened as much as possible, the Torek procedure,[17] directly uniting the tunica albuginea of the testis to the fascia of the thigh, is utilized. This union is severed three months later.

FIG. 8. Retroperitoneal dissection of the cord can be achieved through the same incision by transecting the internal oblique and transversus muscle.[18] The retroperitoneal fat is evident, just medial to the cord. The vas is rarely deficient in length.

In the group of 86 patients operated upon from six to fourteen years of age, two were incarcerated at the time of operation. In three the testis was defective or was not found. Hernia repair was by simple ligation of the sac in most cases. In all cases orchiopexy was performed simultaneously; in three by simple placement of the testis in the scrotum, in thirty-six by the rubber band technic and in forty-four by the Torek procedure. All patients

were followed up to five years; sixty-nine were examined from three months to five years.

RESULTS

In addition to the three defective testes found at surgery, four patients showed poor anatomic results in the follow-up examination. This would indicate seven known unsatisfactory results among the eighty-six patients operated upon in the group of older children, or an incidence of 92 per cent good anatomic results. There were no known recurrent hernias.

From this statistical study it would appear that in those cases of clinical hernia complicated by undescended testis in which surgery is performed from the age of one month to six years, anatomic results as regards the size, consistency and position of the testis when followed up to five years are exceedingly good. A similar conclusion can be drawn from a larger group of children operated upon from the age of six to fourteen years. A comparison of these two groups reveals no advantages as regards anatomic results to be obtained from the postponement of surgery under the conditions of this clinical experience.

SPERMATOGENIC AND ANDROGENIC FUNCTION

The real purpose of surgery in patients with hernia and undescended testis is not only the repair of the hernia and the attainment of anatomically normal testes but also a normally functioning testis, both as regards sperm and androgen. Studies thus far in the latter have been only mildly encouraging. MacCollum[11] did find that in cases of bilateral orchiopexy eleven of fifteen patients were considered fertile and six had had children. However, more recent studies by Hansen[9] showed little or no evidence that orchiopexy performed in mid-childhood, as advocated in the past, increased spermatogenic function. Sperm counts in thirty-six treated and thirty-five untreated unilateral cases were almost identical and averaged about one-half (70,000,000) that which is found in the normal male. Histologic evidence indicates without doubt that after puberty there is obvious deterioration of the spermatogenic tubules. Cooper[5] was of the opinion that this began after the first few years of life but only recently Robinson and Engle[14] established in 111 biopsies that retardation of growth of the spermatogenic tubule begins at about the age of five years and continues as long as the testicle remains in the undescended position. Measurements were equal at an average of 66μ up until the age of five, and from this time on, the diameters of the tubules of the undescended organs lagged behind their mates. Final proof that orchiopexy by the age of five will result in a normal rate of growth has not yet been established but theoretically it is a reasonable assumption and has encouraged surgeons to perform orchiopexy earlier not only in those children with an accompanying hernia but also in all with undescended testis.

It has been practically established by Engberg[7] and others that androgenic function of the undescended testis equals about one-half the normally placed one and that orchiopexy does not improve this deficit.

Thus fertility and histologic studies, and androgenic functional tests indicate that the present orchiopexy performed in mid-childhood or later leaves much to be desired. It is hoped that earlier surgery will improve this situation.

ENDOCRINE THERAPY

Robinson and Engle have introduced a concentrated course of 3,000 to 5,000 units of gonadotropic hormone, given daily for three days, to children between the ages of two and five. They then wait one week; if "spontaneous" descent has not occurred, they operate.

The authors do not advocate this in patients in whom the testicle can be palpated, as the accompanying hernia requires repair and orchiopexy can be easily and safely performed. Thus at present there seems to be no valid reason to subject the child to this added therapy. Palpation usually indicates that the testis lies in the superficial inguinal pouch of Browne,[2] or has passed through the external ring and the cord can be simply lengthened for placement of the testis well in the scrotum.

Palpation is best achieved with the patient standing or crying, so that the orchis can be felt against the tense tendon of the external oblique. If the testis is not felt, it is assumed to lie within the abdomen or the canal. Under these conditions the advantages of any lengthening and enlarging of the structures[6] that follows endocrine administration are obvious to the surgeon.

Endocrine therapy has been utilized in only

a few cases of the combined condition at the Childrens Hospital; but when the testicle cannot be palpated, it appears at the present time to be very useful.

SUMMARY AND CONCLUSIONS

The incidence of hernia complicated by undescended testis in this hospital was 6.7 per cent. There were 134 cases of the combined condition. Forty-eight were operated upon by the age of five; eighty-six were cared for later in childhood. Anatomic results in a follow-up study revealed no advantages in delaying surgery. The surgical technic is described. If the testicle is palpable in the groin, orchiopexy will present no difficulties; if not, a concentrated short course of endocrine therapy seems indicated before surgery.

Most infants and young children with a hernia complicated by undescended testis may have both defects safely and simultaneously operated upon without delay.

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DISCUSSION

Donald T. Hall (Seattle, Wash.): I would like to thank Drs. Chaffin and Snyder for a most enlightening and timely paper. In such commonplace conditions one would assume that the answers were pretty much at hand, and insofar as uncomplicated hernias are concerned I am sure such is true. However, when the factor of cryptorchidism is added, a number of thorny problems arise about which there is a very real field of uncertainty and disagreement. Our own experience in this field has paralleled that of the essayists very closely and we also are now advocating earlier surgical therapy, even in those cases without associated symptomatic hernias. Chorionic gonadotropin in cases of intra-abdominal cryptorchidism, I believe, is debatable, and we have not been too favorably impressed with results in such cases. Certainly, every case of undescended testicle has at least a potential hernia which must be cared for and this is readily done. However, the problem of getting a testicle down into the scrotal sac with sufficient lack of tension that it will stay there is one which I am sure has plagued all of us at one time or another. Again, the difficulty encountered is primarily with those which are high in the canal or in the abdomen. We have had several cases in which, by all known maneuvers, we were unable to get it much below the external ring. I am very much impressed (and I will confess a little envious) by the high percentage of successful cases which Drs. Snyder and Chaffin report (well over 90 per cent). We have had a somewhat larger number of cases of atrophy or of cases in which the final resting place came just below the external ring (prior, of course, to the puberal change). We have been far from satisfied with either the rubber band method or the Torek procedure. We have, in the past few years, come to use more frequently the Ombredanne technic in maintaining and extending the lowermost position accomplished, which simply brings the scrotum up to the testis and

places the latter through the scrotal septum into the contralateral compartment. The constant weight of the scrotum soon brings the testicle down and in time one is unable to detect the fact that the organs are not in separate compartments.

One of the questions which has been raised in this problem of testicular undescent is the character of the testicle itself. Is there a factor of agenesis or dysgenesis which contributes to the failure of descent rather than this being a purely secondary factor? There is some histologic evidence that such is true, in that approximately half of the cases studied have shown some failure of maturation of the seminiferous tubules with a deficiency of the number of Sertoli and Leydig cells, although the correlation of these findings is not sufficiently high to be conclusive. There is much of interest in this field of testicular cytology in cryptorchidism, and I am looking forward with interest to what Dr. Hinman has to say about this in his paper.

The factor of malignancy in the undescended testicle is another undetermined problem. In a recent report by Dr. John Hand of this society who followed a number of cases for a period of many years, there seemed to be evidence that the development of seminoma was likely more related to dysgenesis than to any factor of anatomic location. This is a concept which I predict will be proved true when larger series of cases are followed for greater periods of time.

In conclusion, in light of the evidence at hand I believe, as do Drs. Snyder and Chaffin, that nothing is to be gained by procrastination, for such has been largely the case in the past and results have left much to be desired.

For purposes of interest, in a recent State License Board Examination, I asked a question on this subject. The answers were most revealing, not in the light of knowledge but in the almost total lack of any evidence of consistent teaching concerning this subject.

This paper is most timely, and when one considers the value of the results obtained to the individuals who have their entire lives before them, its importance cannot be suppressed. I should like, again, to thank the essayists for a most worth while paper and for the privilege of discussing it.

WILLIAM H. SNYDER, JR. (closing): I want to thank Dr. Hall very much for his comments. We have used the Torek technic in about half of our cases. It seems applicable in those cases in which, after freeing up the cord as completely as possible and placing the testicle in the scrotum, there is still some retraction. In those cases in which there is real tension we attach the testis to the external ring and allow it to remain in that position for a year or two and then perform a secondary operation similar to a first-stage Torek procedure. This latter technic has been successful in four of the five cases in which it has been used. The fundamental problem which Dr. Hall mentioned is still present with us. No one is certain that the early placement of the testis in the scrotum will result in its normal development and yet placing it early in its proper environment at least offers more hope for normal growth than does a delayed policy.

Endometriosis

ERLE HENRIKSEN, M.D., *Los Angeles, California*

FEW problems in the field of medicine test the diagnostic and therapeutic judgment of the surgeon more fully than endometriosis of the pelvis. Mimicking almost any of the clinical entities common to the lower abdomen and pelvis of the female, it manifests an unusual and unexplainable affinity for the relatively young woman in certain socio-economic groups. It is unfortunate that despite extensive investigations the complex nature of this disease is still not fully understood.

Of the various theories of origin, the retrograde menstruation theory of Sampson and the coelomic metaplasia theory of Iwanoff-Meyer-Novak are the most popular. Although admitting loopholes, as a histopathologist the coelomic metaplasia theory appears to explain all of the various locations, but as a clinician the picture of endometrial fragments fluttering from the end of the tubes is easier to explain.

The term endometriosis implies a condition in which anatomic sites other than the corpus uteri contain tissue possessing the histologic properties of endometrium. Although most of these extrauterine islands also possess the physiologic properties of endometrium, an occasional area does not appear to respond to the cyclic influence of the ovary.

Involvement of the genital organs and the contiguous pelvic structures are by far the most conspicuous. However, the bowel, operative scars and the perineum are not rare sites of this disease. (Fig. 1.) Less common sites are the bladder, umbilicus, kidney, rectus muscles, pectoral fold, and the thigh, arm, lung and pleura. (Fig. 2.)

The incidence of endometriosis is difficult to determine. It is so infrequently observed at the Los Angeles County Hospital as to be considered a clinical rarity. However, the incidence of surgically proved cases in my private practice for the past two years has been 19 per cent. A review of the surgical and pathologic indices of the past twenty years reveals an increase in the incidence of this disease not attributable to an increased awareness of either the surgeon or pathologist.

The material for this article has been collected over a period of twenty years. The study does not represent a consecutive series but a compilation of cases gathered from many sources. Thus an attempt to report organ or site involvement on this disease is impossible. Actually a statistical report on this disease is most difficult because of the accepted error between the diagnosis of the surgeon and the report of the pathologist. This error could be markedly decreased with an improved surgeon-pathologist liaison.

SYMPTOMATOLOGY

A progressive acquired type of dysmenorrhea, dyspareunia, abnormal uterine bleeding and a low backache are the accepted clinical symptoms. However, these cardinal symptoms were present in less than half of the 1,000 records reviewed. Although an acquired type dysmenorrhea is also commonly associated with other lesions of the pelvis, 23 per cent of the patients who were still menstruating had neither dysmenorrhea nor pain. Theoretically the signs and symptoms should reflect the site or sites affected and the degree of tissue involvement, but it is not uncommon to have an extensive pelvic involvement entirely free of pain and a minimal degree of involvement with severe pain.

DIAGNOSIS

The practice of diagnosing endometriosis on the history of a progressive acquired type of dysmenorrhea, despite the absence of correlating pelvic findings, is one of the main reasons for the notoriously low incidence of correct diagnoses. The diagnosis should represent a carefully considered interdependence of the history and rectopelvic examination. Failure to evaluate the uterosacral ligaments, cul-de-sac and rectovaginal septum is evidence of a totally incomplete examination.

Diagnosis of the average case at surgery is not difficult. However, it must be accepted by the surgeon that the pathologic condition, vividly clear at the operating table, loses its

clarity by the time it reaches the pathologist's block. In some instances despite the gross evidence it is difficult to demonstrate endometrial tissue in the wall of the "chocolate cyst of Sampson." The endometrial tissue may be either absent or very scant and the diagnosis

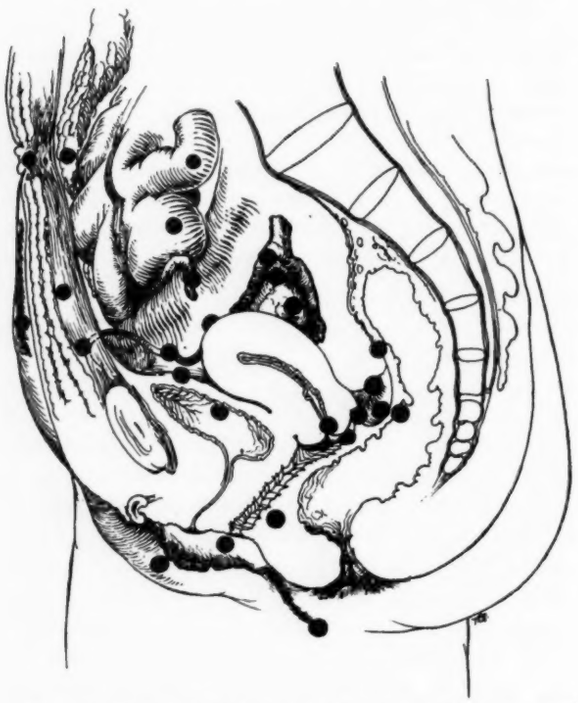

FIG. 1. Sites of endometriosis as noted in 1,000 proved cases.

is based on changes that are not absolute. On the other hand a common error at surgery and the basis of friction with the pathologist, is the surgeon's tendency to consider all "chocolate cysts" of the ovary endometrial in origin.

LOCATION

The usual sites have been described frequently, therefore, the less common locations of endometriosis proved by tissue examination are listed briefly.

Bartholin's gland (one case): A 2 cm. cystic mass in the region of the right Bartholin's gland; tender and possibly enlarged in the premenstruum; no history of trauma or infection.

Vulva (one case): A recurrent, tender, firm nodule on the right labium majus; no pelvic disease at laparotomy; history of trauma twelve years previously at the age of sixteen years.

Vagina (one case): Of the nineteen cases of visible vaginal lesions diagnosed by biopsy, one case was considered of primary origin.

Cervix (two cases): With no evidence of endometriosis of either the vagina or rectovaginal septum, the lesions were considered of primary origin. Both cervices had immediate post-

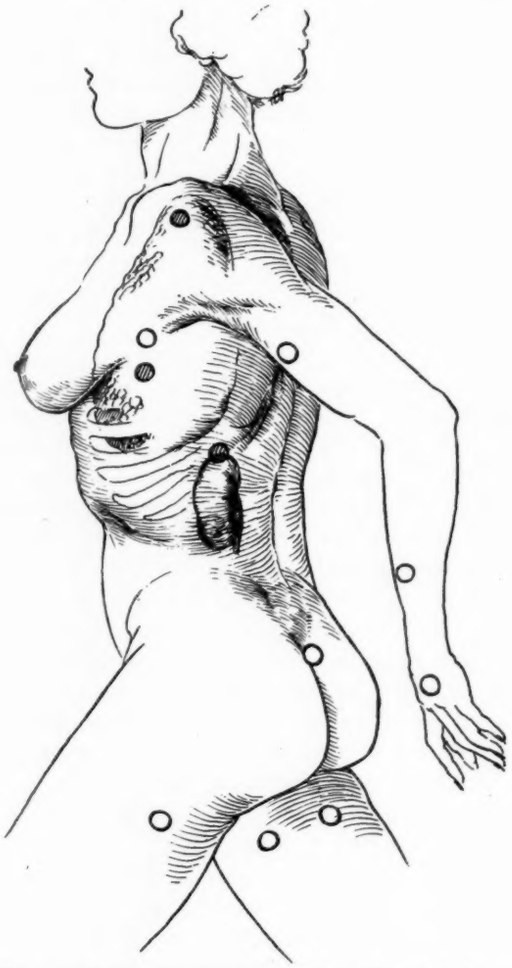

FIG. 2. Less common sites of endometriosis as reported in the literature.

delivery repair. The complaint was postcoital spotting.

Bladder (three cases): Of the three patients with visible evidence of involvement of the bladder mucosa, two were considered as secondary to endometriosis of the pelvis. The third case, with no evidence of pelvic endometriosis, was accepted as of primary origin.[1]

Episiotomy scar (four cases): With all of the aberrant tissue located from 1 to 2 cm. beneath the surface, two of the patients had involvement of the upper angle of the scar: in one case the site was 2 cm. above the anal

orifice; in another of the cases the site was under the hymenal margin. In each instance the subjective symptoms were aggravated in the premenstruum and during menstruation.

Umbilicus (five cases): Three of the patients had definite tumor masses. Two exhibited evidence of infiltration of the surrounding tissue by endometrial tissue and were diagnosed postoperatively. Three of the patients had involvement of the pelvic organs.

Lymph nodes: In a previous report of the necropsy findings in 420 cases of carcinoma of the cervix and uterine corpus, 4 per cent of the treated and 7 per cent of the non-treated patients had lymph nodes containing endometrial tissue.[2]

AGE INCIDENCE

The youngest patient was sixteen years of age and the oldest, eighty-three. Eight of the patients were less than twenty years of age while thirty-seven were in the postmenopausal group. Eighty-two per cent of the patients were in the thirty- to forty-five-year group. The average age was thirty-seven years. In studying the age groups affected, the frequency of endometriosis in relatively young women emphasizes the tragic seriousness of this disease.

THE POSTMENOPAUSAL CASE

As a rule endometriosis tends to regress to a state of little or no importance following the menopause. That this is fortunately true in most instances is evidenced by the characteristic regression of the lesion following surgical castration or menopause. However, exceptions deserve comment. Of the twenty-nine cases in which the complete removal of the ovaries was proved by examination of the surgical specimen, evidence of continued activity of the disease was corroborated by both surgery and tissue study. Various explanations for this failure of tissue regression have been advanced, namely, incomplete removal of the ovaries, a third or aberrant ovary, or an extrapelvic origin of estrogen.

With two years of amenorrhea as an acceptable time interval, the records of thirty-seven patients in the postmenopausal period were reviewed. The youngest patient was forty-four years of age and the oldest, eighty-three. In six cases the diagnosis was made by the pathologist following radical surgery of the large bowel for suspected neoplasm. In four cases pelvic endometriosis was incidental to proved cancer of the bowel or rectum. Whether the malignancy was of endometrial origin was questionable in one case. In three cases endometriosis was found during surgery for cancer of the uterine corpus. In one case endometriosis involved one ovary, whereas the contralateral ovary contained a granulosa cell tumor. In the remaining twenty-three cases a history of persistent lower abdominal and pelvic distress, usually of many years' duration, eventually brought the patient to surgery. The possibility of endometriosis was not considered in any of these cases.

BOWEL

Endometriosis usually considered a problem of the pelvis, merits an important position as a benign lesion of both the small and large bowel. Although rarely possessing a malignant potentiality, it frequently exhibits such infiltrative tendencies as to make it rather easily confused with a malignant neoplasm. This is especially true when the bowel involvement is so extensive as to cause signs of obstruction.

Endometriosis of the appendix (seventeen cases) is briefly mentioned to emphasize an important point in the diagnosis and treatment of this disease. The McBurney incision was employed in the removal of five appendices with the diagnosis of "chronic appendicitis" and two with the diagnosis of "regional ileitis." In only one of the seven cases was an attempt made to examine the pelvic organs. Although it is admitted that the McBurney incision has its merits, it is evident that the incision does not permit an adequate evaluation of the pelvic organs, an invaluable procedure in the female patient with the vague diagnosis of "chronic appendicitis."

Small Bowel (Twenty-eight Cases). Any portion of the intestinal tract which lies in or near the pelvic cavity may be affected by this disease. (Fig. 3.) In all of the twenty-eight patients with involvement of the small bowel, endometriosis was present in the pelvic area. In seventeen cases the implants were incidental and asymptomatic. In most instances the implants were located on the distal portion of the ileum, although in two cases multiple implants extended to a distance of 40 cm. The lesion is usually limited to serosa and tends to remain asymptomatic unless the muscularis

and, less frequently, the mucosa are involved. In two cases the presence of melena was ascribed to mucosal penetration.

The preoperative diagnosis in the eleven patients with signs of probable disease in the right abdomen included appendicitis, ileitis,

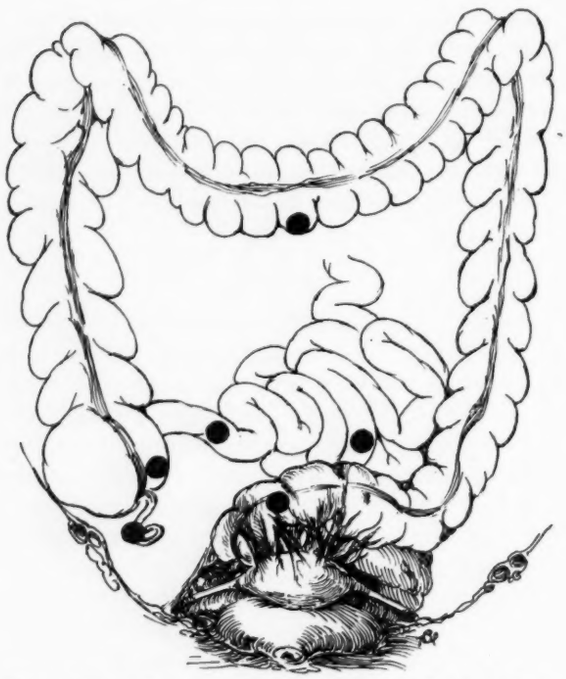

FIG. 3. Locations of endometriosis of the bowel reported in this series.

neoplasm, postoperative adhesions and tuberculosis. In the seven patients with definite obstruction the site of the obstruction was localized by roentgengrams. In nine of the eleven cases the subjective signs of obstruction were aggravated in the premenstruum. However, a study of patients without endometriosis, but in the same age group, also exhibited a tendency to symptom aggravation in the premenstruum or during the period.

Of the four patients with the picture of an acute small bowel obstruction, one patient presented the so-called typical picture of progressive obstruction on a cyclic basis.

It is most important to be aware of the possibility of endometriosis as a factor in problems involving the small bowel. Of equal import is adequate examination of the pelvis and its contents whenever the female abdomen is opened. In our experience the absence of the disease in the pelvis makes the diagnosis of endometriosis of the small bowel unlikely.

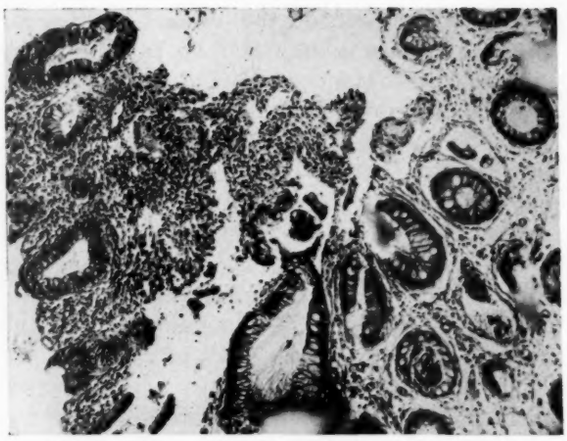

FIG. 4. Biopsy of endometriosis of the rectum.

Large Bowel and Rectum. Involvement of the large bowel and the rectum are relatively frequent and the cases herein reviewed do not represent the probable incidence. Except for two cases all were considered of secondary origin to endometriosis of the ovary, cul-de-sac or recto-vaginal septum. Of twenty-seven proved cases of endometriosis of the recto-vaginal septum, the bowel or rectum was involved in twenty-one. The two cases considered of primary origin included a fifty-five year old woman, four years postmenopausal, with a large irregular mass of ligneous consistency involving the sigmoid from the 12th to 22nd cm. level. The mass was freely moveable. Despite the history of profuse rectal bleeding, no evidence of mucosal penetration could be visualized. The second case was a thirty-eight year old woman presenting with painful, bloody stools and a low backache which were aggravated in the premenstruum and during the period. The diagnosis of endometriosis was proved by biopsy of the lesion at the 14 cm. level. (Fig. 4.) Complete recovery of both cases followed the removal of the ovaries. No other viedence of endometriosis was found.

Rectal bleeding was the primary complaint of nineteen patients. Perforation of the mucosa was demonstrated in four of this group. In most instances of rectal bleeding the source was not determined. However, it is probable that the mucosa may be involved but the site is not readily located. Examination of the large bowel and rectum in these cases during the period added nothing. On visual examination the positive findings included a narrowing of the bowel lumen, but this evidence of constriction may be secondary to the pressure of a

large ovarian mass. Occasionally the high location of the constriction or distortion secondary to pelvic adhesions make visualization impossible. Roentgen studies have revealed no characteristic pattern that would assist in the diagnosis. Unlike the lesion of the small bowel endometriosis of the large bowel and the rectum frequently present a suggestive clinical picture consistent with the site involvement. Fixation of the upper vagina and cul-de-sac, and the impingement and distortion of the bowel often cause dyspareunia, dyschizia, occasional rectal bleeding and various degrees of obstruction. Complete obstruction however is uncommon. In most instances symptoms are aggravated in the premenstruum and/or during the period.

ACUTE CONDITION OF THE ABDOMEN

Endometriosis was considered the basis of an acute abdominal condition in nine cases. In eight patients a spontaneous rupture of an ovarian cyst was present, followed by the typical picture of an acute peritonitis. The size of the cysts was not determined. Of surgical interest was the accidental opening of the bowel in five of the cases. There were two deaths, and these were considered of surgical origin.

In one case a perforation of the sigmoid was interpreted preoperatively as rupture of a diverticulum. At surgery a radical resection was performed with the diagnosis of extensive cancer. The correct diagnosis was made at autopsy.

Episodes of less severe character probably are more frequent than this review suggests.

The usual extensive involvement of the pelvic organs and the tendency of both the small and large bowel to adhere to the pelvic organs by hard, dense adhesions all add to a difficult operative field for the most experienced surgeon.

TREATMENT

Treatment is dependent on the symptoms, palpable and visual extent of the disease, age and general physical condition of the patient, the need and desire for children, and the psychosomatic evaluation of the patient. The problem of endometriosis is not restricted to the gynecologist and treatment requires more than the technical mastery of the scapel. Proper management requires a thorough understanding of the potential seriousness of the disease, the indications for and limitations of surgery, and the probable right and possible wrong approach. Therapeutic enthusiasts, be they conservative or radical, tend to follow a road that permits little individualization, an approach of utmost value in the problem of endometriosis.

Fortunately the trend toward conservative management of endometriosis is becoming more widely accepted. Conservative management implies preservation of the ability to bear children. In many instances endometriosis is a probable preoperative diagnosis and the patient should be made completely aware of the possible minimal and maximal scope of the planned surgery. The possibility of future operations, if surgery is conservative, and the ability to remain a normal active woman, if surgery is radical, are both stressed. The young woman with endometriosis deserves every chance; however, there is always the danger that conservatism can be carried to extreme.

Medical treatment of endometriosis has not been too effectual in our hands. Accepting the frequency of error in the diagnosis, the statistical evaluation of any form of hormonal therapy must be accepted with skepticism. However, the carefully planned use of androgens has been of occasional value in those patients seeking temporary relief. We have had no experience with large amounts of estrogens.

Although the use of estrogens following radical treatment of endometriosis has long been considered ill advised, it is our opinion that there is no reason for withholding carefully supervised estrogen therapy from patients with disturbing menopausal symptoms.

Conservative surgery should be considered in the younger patient, desirous of children, in whom the extent of the disease permits the preservation of the essential organs. Of added value to the conservative approach is the resection of the superior hypogastric plexus. Although this operation will not affect the pain of ovarian origin, it will often prove invaluable in the management of uterine dysmenorrhea. Radical surgery should be considered in the older patient or in the patient in whom involvement is so extensive as to prevent the preservation of the essential organs. Thus it is apparent that the management of the disease requires strict individualization.

Massive infiltration of the large bowel, unless obstruction is an acute problem, responds to the

removal of the ovarian tissue. The regression of the bowel lesion is gradual and the residue rarely causes any discomfort. In rare instances of obstruction, colostomy may be indicated. However, there is no longer any justification for the extensive and hazardous resections originally advocated by Cullen. When operating in a pelvis which is extensively involved, it is well to remember the maxim that it is better to leave a bit of the uterus on the bowel than a bit of the bowel on the uterus.

Surgery for lesions of the small bowel are rarely indicated unless obstruction is present or the involvement so extensive as to increase the possibility of a later complication.

SUMMARY

1. Observations based on review of 1,000 cases of proved endometriosis are presented.

2. The so-called characteristic sign-symptom pattern was present in 43 per cent of the cases.

3. The disease is diagnosed on an awareness of the possibility of the disease, a careful history and a thorough rectopelvic examination.

4. Although the disease tends to regress following surgical or physiologic castration, twenty-nine patients exhibited clinical and histologic evidence of continued activity following ovariectomy. Active endometriosis was proved in thirty-seven patients in the postmenopausal group.

5. Involvement of the bowel is frequent enough to warrant the acceptance of endometriosis as an important possible factor in the problems affecting both the small and large bowel.

6. An acute condition of the abdomen followed the spontaneous rupture of an ovarian cyst of endometrial origin in eight cases; a perforation of an extensive endometrioid lesion of the sigmoid in one case.

7. Proper management is based on the surgeon's appreciation of the natural history of the disease, the evaluation of such factors as age, severity of symptoms, extent of disease, desire for children, and the patient as a whole. Fortunately the value of conservatism in the surgical management of the disease is becoming more widely accepted.

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DISCUSSION

Howard C. Stearns (Portland, Ore.): This review of endometriosis given by Dr. Henriksen comprises an unusually large number of carefully reviewed, proved cases. It is of real value to me in many ways. While it confirms my general belief in regard to symptoms, or the lack of symptoms, diagnosis, management and so on, I was surprised to learn of the frequency of small bowel involvement found in his series. I have seen a few such tiny, apparently rather inactive lesions on the ileum, but only one of marked extension. In this case, a thirty-six year old woman suffered marked endometrial involvement of the pelvic viscera. At approximately 20 cm. superior to the cecum a gross lesion of the ileum was found, resembling an endometrioma. The bowel was approaching complete obstruction, with marked dilation superior to the lesion. Above the point of near obstruction a firm, flattened foreign body could be palpated. The lesion was removed and the bowel anastomosed end-to-end. The foreign body appeared to be a piece of synthetic floor covering such as asphalt tile, and measured roughly 3 cm. by 5 cm. in dimension. How it passed the esophagus or how it got there seemed as much a mystery to the patient as to me.

Dr. Henriksen deserves commendation for his attitude regarding preservation of childbearing possibilities in young women, and of ovarian physiology wherever reasonable in women under the menopausal age. In a series of 139 proved cases of endometriosis previously reported by me, ovarian tissue was allowed to remain in 84.3 per cent with only a few failures among them. I heartily endorse Dr. Henriksen's attitude and also his views on individualization in management of this rather bizarre disease.

Of particular interest to surgeons at this time is the problem of the young person afflicted with a fixed, tough, deep lesion in the cul-de-sac, involving the tissues of that area and deeply invading the anterior rectal wall at and beneath the peritoneal reflexion. Such a person is usually infertile. She often reveals no other endometriomas. It is my opinion that if such lesions could be totally resected, many such women would then achieve pregnancy. Most general surgeons with whom I have discussed such problems decry partial bowel resection in such an area. Personally I would appreciate comment on this problem from Dr. Henriksen and others.

Many questions regarding endometriosis remain unanswered. The etiology stems apparently from more than one factor. Certainly metastatic and

surgical transplants are often apparent. Yet the theory of Sampson leaves much to be answered. In my experience the disease is more commonly found in the patient with an anteverted uterus than in one with retroversion (only thirty-five of 139 women in my series had uterine retroversion). Why do patients with endometriosis have low fertility? Is some substance toxic to the liberated ovum exuded from the lesion? When such lesions are found and extirpated in the infertile woman, pregnancy often follows. Why is the disease uncommonly seen in the charity hospital, yet abundantly plentiful in the private hospital? Early marriage of the indigent group as compared to the financially superior group appears not to be the complete answer.

In conclusion I wish to thank Dr. Henriksen for supplying us with such an abundance of reliable statistics on this malady. Anyone who has reported studies of even 100 such cases will appreciate the time, toil and tribulation involved in the proper review of 1,000 cases.

WILLIAM B. HUTCHISON (Seattle, Wash.): I simply rise to cite a case which occurred in Yakima, Washington. It was not a case of mine. This was a young lady who had an endometrial implant in the apex of her left lung. The diagnosis was made preoperatively on the strength of a history which was perfectly typical. The woman had hemoptysis which occurred at the time of her menstrual cycle on three different occasions. X-ray revealed an endometrioma at the left apex. A segmental resection was performed and it was proved at pathologic examination to be an endometrial implant. The woman made an uneventful recovery.

ERLE HENRIKSEN (closing): May I commend the conservative management of endometriosis by Dr. Stearns. His 84 per cent conservative approach overwhelms my probable 45 per cent. Also, I would like to restate that the 1,000 cases in this series do not represent my own cases. In the past twenty years I have had 209 cases which clinically and pathologically warranted inclusion in this group. Regarding the advisability of bowel resection it takes little experience to make general statements, but it takes experience tempered with judgment to deal with individual problems. No disease in the field of gynecology demands as strict individualization as does the management of endometriosis. From my experience the resection of the small or large bowel, in the presence of the disease elsewhere, will in most instances suggest faulty surgical judgment. There is a tendency on the part of the surgeon to diagnose endometriosis and, when the abdomen is opened, to ease his conscience with the findings of a small inactive endometrioid lesion in the cul-de-sac. No tissue is sent to the pathologist, who is then accused of being both inefficient and uncooperative. Regarding endometriosis of the pleura I can say that hemoptysis in the premenstruum and during the period is not rare. Two or three times each year I am consulted regarding the diagnosis of endometriosis of the lung based solely on the history. This, of course, is not possible.

In endometriosis we have a disease of serious potential, characterized by misdiagnosis and mismanagement. No disease affecting the female can be properly managed unless its natural history is understood and the therapeutic modus operandi is conscientiously and intelligently appreciated.

Cancer of the Thyroid in Children

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THE increasingly important problem of malignant goiter was demonstrated by Searls in his recent studies on the incidence of

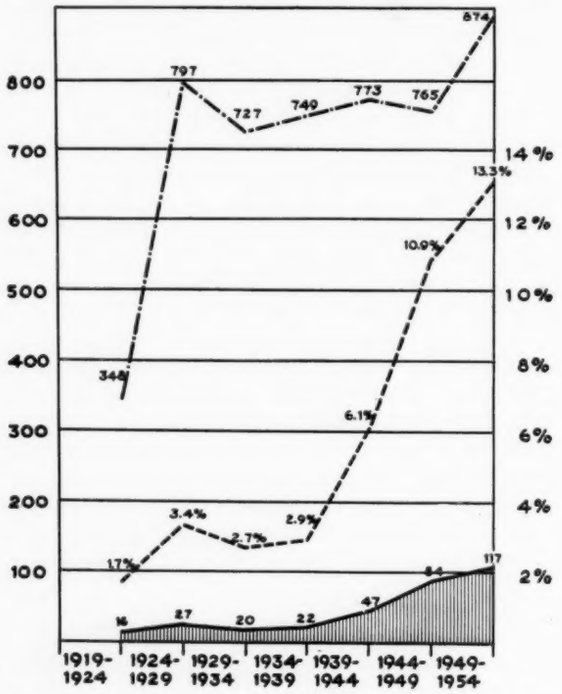


FIG. 1. Percentage of malignancies in nodular goiter at the University of California Hospital computed by five-year periods from 1919 to 1954. (Key: —— five-year total of thyroidectomies for nodular goiter; - - - per cent ratios of malignancy; ——— number of malignancies encountered in these totals.)

the disease observed at the University of California Hospital.[1] He showed that in the last two decades the percentage of malignancy in specimens removed for nodular goiter rose from 2.9 per cent in the five-year period from 1934 to 1939 to 13.3 per cent in the five years from 1949 to 1954. (Fig. 1.) That the problem is even more serious in children is shown by Figure 2 which demonstrates that malignancy exists in 47 per cent of the nodular goiters removed in patients from birth to age fifteen, and in 32 per cent of nodular goiters up to and including the age of twenty-one years.

Because they are typical of cancer of the thyroid in children, the following three cases are reported in detail. These patients have been followed long enough to furnish a clue as to how similar cases may respond.

	ALL GOITERS	NODULAR GOITERS
ALL AGES	4.0	7.0
AGE 0 - 15 INCLUSIVE	17.5	47.0
AGE 0 - 21 INCLUSIVE	9.3	32.2

FIG. 2. Percentage of malignancies in specimens removed at operation for goiters at the University of California Hospital from 1912 through August, 1954.

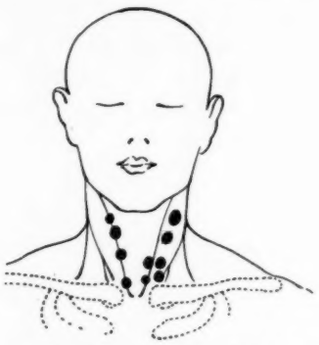


FIG. 3. Case 1. Distribution of nodules at operation in July, 1931.

CASE REPORTS

CASE 1. J. R. E., a nine year old boy, came under medical observation in July, 1931, for hyperthyroidism. He received ten treatments by x-ray in the six months from July, 1931 to January, 1932, calculated at 1,000 r at 5 cm. depth. With this treatment his metabolism dropped from 42 per cent plus to 16 per cent plus.

In June, 1932, at the age of ten, he was found to have multiple nodules along both sterno-

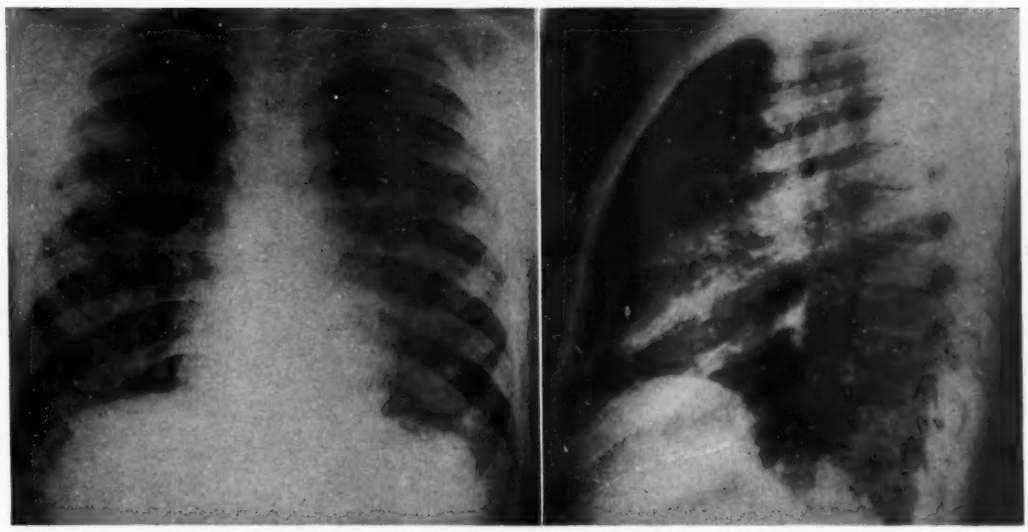


FIG. 4. Case 1. Chest metastases at time of operation in June, 1932.

mastoid muscles as well as in the thyroid gland. (Fig. 3.) Complete resection of the left lobe of the thyroid gland and dissection of the left cervical lymph nodes was immediately performed. Pathologic studies of tissue removed at operation showed adenocarcinoma of the thyroid. (Fig. 5.) X-rays of the chest were positive for metastases from the year 1932 onward (Fig. 4), but in spite of these pulmonary metastases the patient remained in relatively good health.

In 1940, when the patient was eighteen years of age, the cervical nodes began to enlarge. The growth in these nodes was slow. In June, 1944, he received nine x-ray treatments in eleven days to the left cervical nodes, with a calculated dose of 2,000 r at 2 cm. depth. The following month over an eleven-day period he received ten treatments intraorally, calculated at 750 r at 2 cm. depth. Both the intraoral lesion and the cervical node responded well to this therapy.

In January, 1947, the tumor was found to absorb radioactive iodine (I-131) in amounts large enough to encourage its therapeutic use. Radioautograph of the biopsy material is shown in Figure 6. Because I-131 was a new therapeutic weapon at that time (1947), the patient was given only 110 mc.

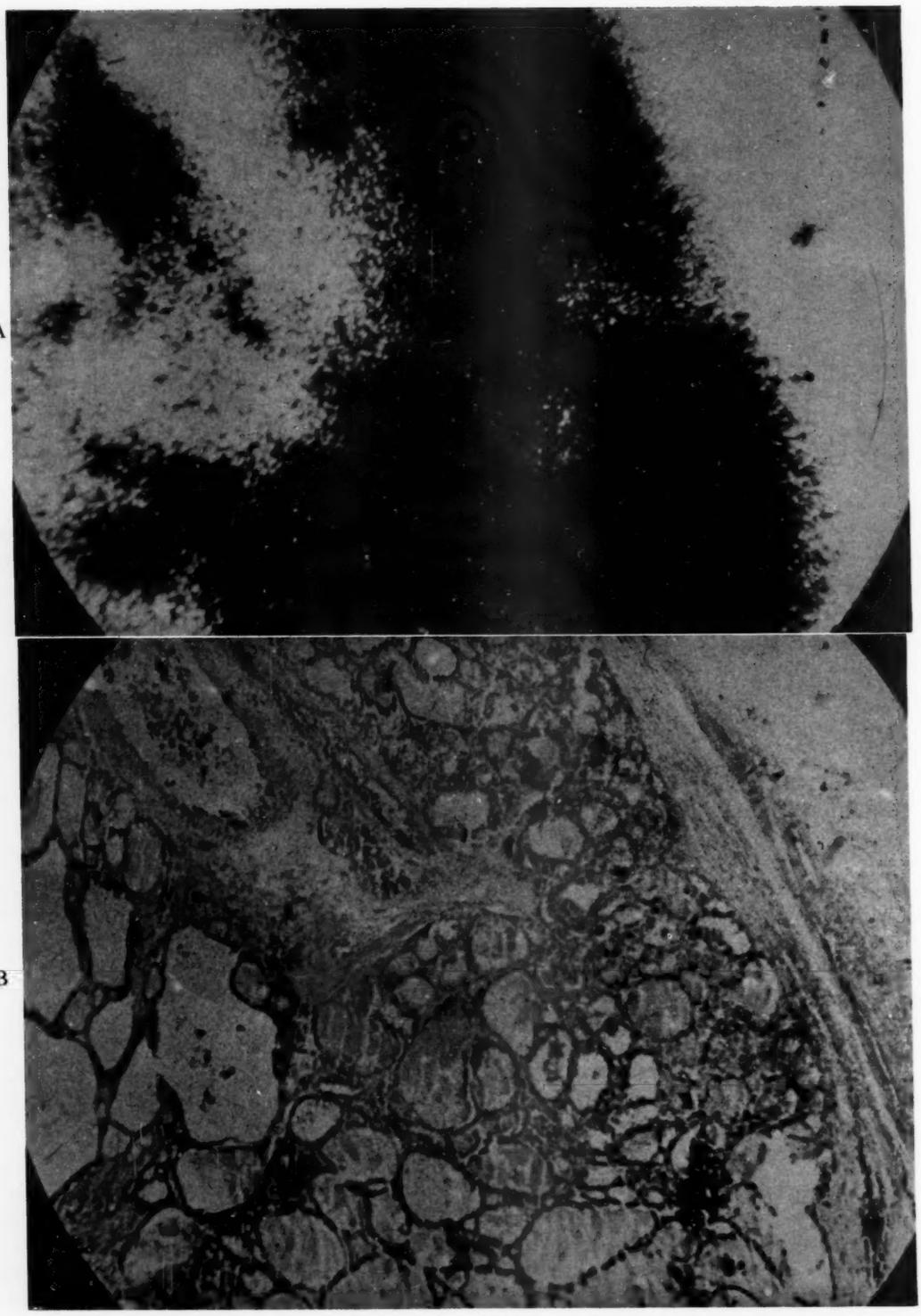
By 1949 there was a better understanding of radioactive iodine; and because a fixed vocal cord and apparent infiltration of tumor along the left pharyngeal wall had developed, the patient was given 337.5 mc. of I-131 over a three-month period. The therapeutic effect

FIG. 5. Case 1. Adenocarcinoma. Photomicrograph of operative specimen (June, 1932).

was so satisfactory that all evidence of pulmonary and cervical metastases, so far as could be determined by clinical examination, disappeared. (Fig. 7.) In keeping with the apparent disappearance of pulmonary metastases as shown on the x-ray film, tracer doses of I-131 were no longer absorbed into the lungs in amounts which could be measured with the scanning apparatus (Geiger counter).

In 1950 tumor activity in the paratracheal region caused bilateral vocal cord paralysis, and in March of that year a King operation was performed, displacing one arytenoid cartilage laterally and fixing it so as to give an adequate airway.

In September, 1952, biopsy of the neck metastases showed an anaplastic type of



6A

6B

FIG. 6. Case 1. Section of tissue in biopsy specimen, demonstrating extensive uptake of radioactive iodine. A, radio-autograph. B, photomicrograph of same section. (From the Radiologic Laboratory of the University of California School of Medicine, A.E.C. Contract No. AP-11-1-GEN-10, Project No. 2.)

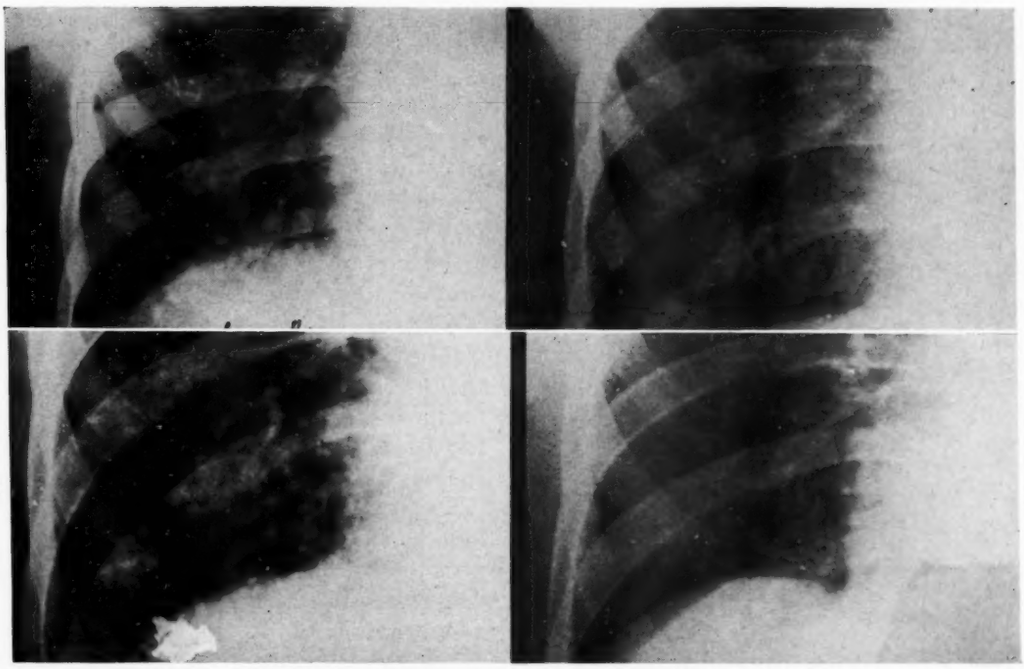
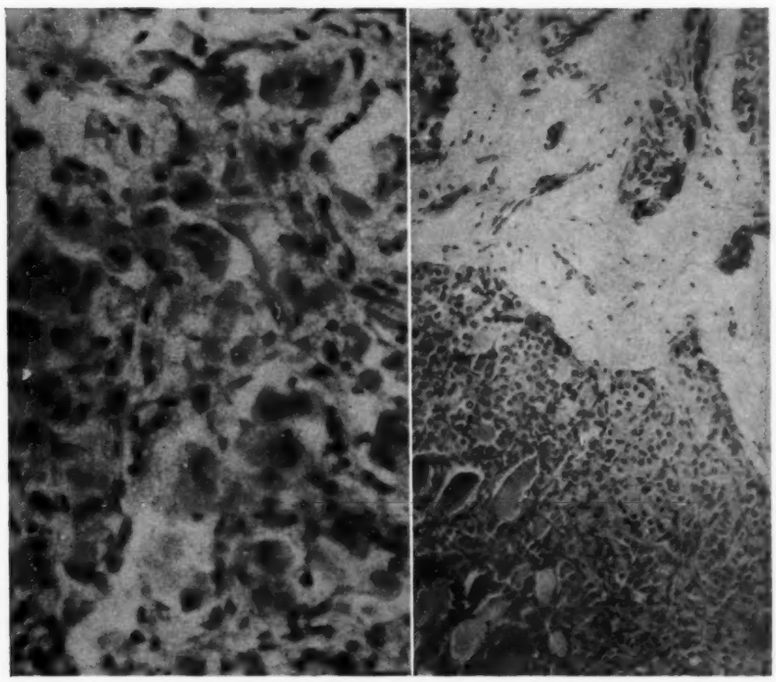


FIG. 7. Case 1. Progress of pulmonary metastases. A, 1932. B, 1937. C, 1947. D, 1952, apparent disappearance following therapeutic use of radioactive iodine.



8A 8B

FIG. 8. Case 1. A, microscopic view of biopsy specimen (September, 1952) demonstrating the anaplastic type of the recurrent growth as compared with (B) the original lesion.

growth in the supraclavicular region. (Fig. 8.) Tests with I-131 had demonstrated a very poor uptake in July and August, 1952, and the anaplastic nature of the growth appeared to be an explanation for this lack of iodine absorption. The uptake of I-131 could not be

FIG. 9. Case II. Distribution of neck nodules at operation on September 24, 1930.

enhanced by administration of thyroid-stimulating hormone.

The patient died on October 16, 1952, at the age of thirty. The autopsy examination showed the following: (1) adenocarcinoma of the thyroid with metastases to lungs and pleura, lymph nodes of mediastinum and abdomen, left adrenal and kidney, and tissues of the neck and trachea; (2) obliteration of the thyroid gland by surgery and radiation; and (3) pulmonary embolus from the left leg.

Among the interesting and unusual features of this case is the fact that pulmonary metastases were present from the age of ten, when the patient was first operated upon, until the age of eighteen, without giving any clinical evidence of their existence and without interfering with the patient's progress through grammar school, high school and college. There was nothing but the evidence of x-ray to show that his lungs were studded with metastases.

The second remarkable feature of this case was the unusual affinity of the malignant tissue for iodine. These were the years when I-131 was first available for clinical study, and the patient, himself a physicist, was one of the anxious observers who watched the activity on the Geiger counter with his physicians.

The third notable feature of this case was the anaplastic nature of the recurrences which took place after effective use of I-131 as a therapeutic agent. These anaplastic growths, when they recurred, could not be induced to absorb radioactive iodine again and quickly brought about the death of the patient.

CASE II. J. W., a sixteen year old girl, was seen in August, 1930. Before her first visit to us she had had a mass removed from the left posterior cervical triangle. (Fig. 9.) This mass had been diagnosed as lateral aberrant thyroid tissue. The patient was operated upon on September 24, 1930, at which time right partial lobectomy, left total lobectomy and left radical neck dissection were performed. The pathologic diagnosis of tissue removed at operation was adenocarcinoma of the thyroid with lateral cervical metastases. (Fig. 10.)

Following operation this patient received x-ray therapy, totalling 2,500 r, from September 1930 to January 1931.

She has been followed at one- to two-year intervals and has remained well without recurrence until the present time. She is now forty years old and the mother of four normal, healthy children. The follow-up period has been twenty-five years, and it seems unlikely that a recurrence will take place now when it has not done so for this length of time.

CASE III. C. L. B., a baby girl, was born December 30, 1932. In January, 1936, at three years of age, a mass described by her pediatrician as being 4 cm. in diameter was found in the left lobe of the thyroid gland, and palpably enlarged lymph nodes were present elsewhere in the neck. These developed rapidly, so that by April, 1937, when the patient was four and a half years old, her condition was as shown in Figure 11. Biopsy of one of the larger nodes at that time showed papillary adenocarcinoma. (Fig. 12.)

This patient received radiation therapy with 200 kv. generated x-rays. The quality of the radiation was equivalent to a half value layer (H.V.L.) of 2 mm. copper. The treatment was given to the neck through four ports (anterior, posterior, right and left lateral). Each field size was 10 by 10 cm. Twenty-four treatments were given in thirty-one days. The total tissue dose calculated in the mid-plane center of the

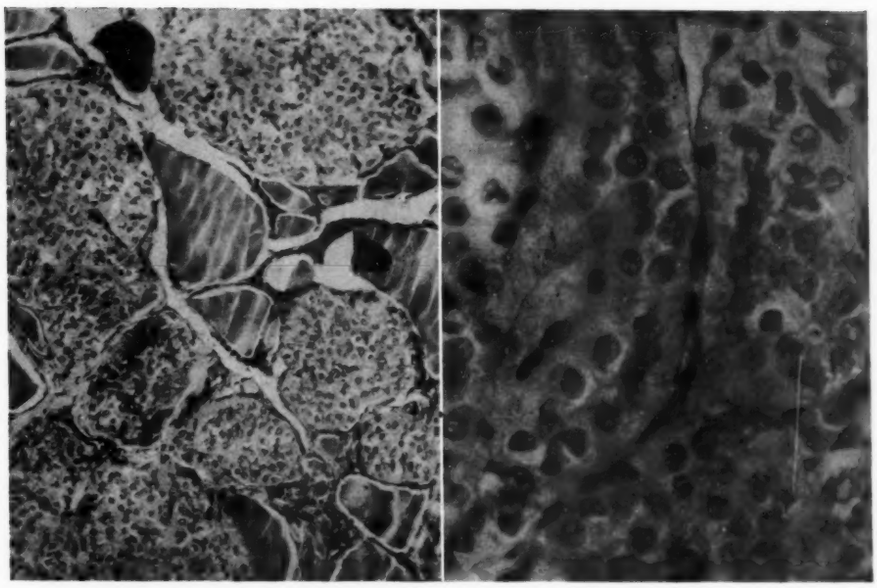


FIG. 10. Case II. Photomicrographs of specimen (September, 1930).

FIG. 11. Case III. Patient at four and a half years of age; after biopsy but before x-ray therapy.

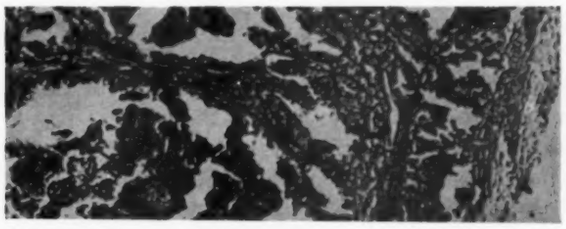


FIG. 12. Case III. Photomicrograph of biopsy specimen. Papillary carcinoma. Each succeeding operative specimen showed an identical picture.

neck was approximately 2,900 r. Each field received approximately 900 r (air). Thyroid substance was given in tolerance doses, as recommended by Sir Thomas Dunhill. The tumors disappeared and the patient remained well for the next several years.

Early in 1947 the patient, then fourteen years old, neglected taking thyroid substance regularly, and the tumors began to grow in number and size. She was given a test dose of radioactive iodine. Radioautograph of a biopsy specimen taken forty-eight hours after administration of the test dose revealed no absorption. Total thyroidectomy and bilateral neck dissection were performed. Pathologic examination showed a picture identical with that of 1937, and the pathologic diagnosis was papillary adenocarcinoma with invasion of (1) capsule, (2) blood vessels and (3) cervical lymph nodes. Following this operation clinical examination showed one small nodule remaining just below the left mastoid process.

From 1947 to 1953 the patient's health remained excellent, and she took thyroid substance, 0.13 gm. daily, until December, 1953. At that time she was returned to surgery and the remaining nodule, palpable beneath the left mastoid process was removed. The pathologic picture was the same as it had been in 1937 and 1947.

The patient has remained well with no clinical signs of recurrence up to the time of this report. She is now twenty-two years of age, and the follow-up period has been eighteen years.

One of the striking features of this case is the apparently excellent response to radiation therapy and the apparent dormancy of the

tumor over a period of thirteen years while the patient was taking thyroid substance. The second most interesting phase was the apparent stimulus to growth when the patient neglected to take thyroid substance, leading to the radical operation of 1947. The tumor then remained quiescent while the patient was taking 2 gr. of thyroid substance regularly. The third and probably most interesting feature of the case is that the tumor removed in December, 1953, showed a pathologic picture identical to that of the original biopsy performed seventeen years earlier when the patient was four and a half years old. Evidently the prognosis will continue to be good so long as the tumor remains of the predominantly papillary type.

CONCLUSIONS

These three cases represent typical examples of childhood carcinoma of the thyroid. They suggest that therapy must be individualized according to the pathologic picture, the possibility of complete extirpation, the presence of metastases, the affinity of the tumor for radioactive iodine (I-131), and the sensitivity of the tumor to external radiation. The prognosis will depend largely upon these factors in each case.

If the outcome in these three patients can be used as a guide in similar cases, certain conclusions seem justifiable. The first is that pulmonary metastases may exist for years without apparently affecting the health of the patient. Two of the cases demonstrate that long coexistence with cancer of the thyroid having local and even pulmonary metastases is possible with very little evidence of adverse effect on the health of the individual.

Second, if distant metastases are present, they are of grave eventual import, even if affinity for I-131 is shown by these deposits. Recurrence after therapeutic radioactive iodine is particularly serious if the recurrent growth is more anaplastic and shows less tendency to absorb radioactive iodine.

The third conclusion is that a long survival and possibly a cure can be expected in cases of childhood cancer of the thyroid, even after lateral cervical metastases have taken place, if the tumor is of a papillary pattern or if it is sensitive to external radiation.

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Nodular Goiters in Children

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GOITERS in children can be classified into two principal groups: (1) congenital and (2) those which appear later in childhood. About seventy cases of congenital goiter have been seen in North America and of these seventeen had some type of surgical treatment. Congenital goiter is a sporadic disease and there is no region in America in which it is truly endemic. Only one case has been previously reported from Los Angeles.[14] There have been three more at the Children's Hospital, one of them treated surgically. The vital statistics for the city of Los Angeles show that there has not been a death from congenital goiter in the last ten years.

Many normal children of both sexes have been born before and after the birth of siblings with congenital goiter. These babies have been born to normal parents living in goiter-endemic areas and to goitrous parents living in non-endemic areas. We have been unable to relate congenital goiter to the intake of iodine, antithyroid drugs or thyroid by the mother during pregnancy.[6,7,9,16,23,24,26,29,30,34,35,41,44]

The clinical features of congenital goiter are of surgical interest. The diagnosis can easily be missed, the confusion arising from three facts: (1) Congenital goiters almost invariably appear as a very soft, almost "cystic" tumor. Attempts have been made to transilluminate them.[4,14,25,32,36] (2) Several authors mention the "unusually" high position of the gland with the upper poles almost in contact with the mandible bilaterally. Substernal extension is almost unknown.[1,6,25,28,32,40] (3) The most striking feature is that these goiters extend *behind* the pharynx and trachea. Some large goiters can be palpated through the posterior pharynx. Lateral soft tissue x-rays of the neck will often demonstrate *anterior* displacement of the pharynx and trachea. In many reports the goiter "encircled" the pharynx and the lateral lobes were almost in contact posteriorly.[1,4,6,16,22,24,25,32,36] Typically these infants lie with the head retracted and the respiratory distress is aggravated by flexing the neck.

The treatment of congenital goiter includes five possibilities:

1. Some of them clear completely without any treatment.[17,23,35]

2. Iodine by mouth has been used but failed to reduce the goiter in many cases. The rubbing of iodine ointment into the skin has now been abandoned. In some cases the use of iodine confused the diagnosis of hypothyroidism and delayed effective treatment with thyroid.[26,30,44,45]

3. The use of thyroid to reduce congenital goiters has been more successful. Rienhoff gave thyroxine hypodermically in one case with prompt improvement. The consensus of opinion is that thyroid is preferable to iodine if medical treatment is to be attempted.

4. X-ray therapy has been used in two cases with no benefit. By a peculiar coincidence both of these had Hürthle cell tumors, one of which was malignant.[25,31]

5. The evidence suggests that surgery has not been used often enough. At least ten patients have died of untreated respiratory obstruction due to congenital goiter.[1,6,14,23,24,30,35,37] The results from thyroidectomy have been uniformly excellent. Every one of the babies treated by thyroidectomy was relieved of the respiratory obstruction and survived.[4,6,25,28,30,32,33,40,45] In contrast, two babies treated by tracheotomy died,[19,34] and a third, in whom thyroidectomy was recommended but was refused by the parents, was treated by tracheotomy and also died.[22] In one family the death of two babies from congenital goiter led their doctor to take a firm stand about surgery for the seventh child. Thyroidectomy was performed and the baby survived.[6] In one case hemithyroidectomy did not relieve the obstruction and the other lobe had to be removed one month later.[4] If a baby with congenital goiter has respiratory obstruction and the goiter extends behind the pharynx and trachea, the treatment of choice is thyroidectomy.

There is a real hazard of malignancy, even at this early age. Five patients with cancer in a goiter present from birth have been re-

ported. One was operated upon at three, one at four and one at seven years of age.[46] Another patient had four operations from the age of six months to eleven years and finally died of cancer at the age of sixteen.[33] The fifth case of cancer of the thyroid is the earliest found in the literature, a baby girl operated upon at one month of age after x-ray therapy had failed to reduce the size of a congenital goiter. Hürthle cell adenocarcinoma was found, and in spite of postoperative x-ray therapy the baby died one year later of recurrent cancer.[31]

TABLE 1
NODULAR GOITERS IN CHILDREN
(Solitary Non-toxic Nodules)

Patient	Sex and Age	History	Pathology
D. G.	F, 5	3 yr.	Fetal adenoma
B. K.	M, 11	4 yr.	Fetal adenoma
S. L.	F, 13	1 mo.	Fetal adenoma (D. H. Sprong)
M. S.	F, 10	8 yr.	Colloid adenoma
L. F.	F, 3	2½ mo.	Colloid adenoma
R. M.	M, 11	8 mo.	Adenoma, fibrosis
J. S.	M, 4	2 yr.	Colloid adenoma, 1948; recurrent nodule since 1951
P. L.	F, 4	From birth	Colloid adenoma, 1950; pyramidal lobe recurrence; colloid adenoma, 1951
E. C.	F, 9	Toxic diffuse goiter; hyperplasia, marked, 1948; three-stage operation; thiouracil, 1 yr.	Adenoma, cystic degeneration, 1951
J. Y.	F, 6	Died (subacute bacterial endocarditis)	Adenoma (autopsy)

At the Children's Hospital in Los Angeles we have had fourteen patients with non-toxic nodular goiters in the last eighteen years; ten were girls, four were boys. The age range was three to thirteen years. One adenoma was found at autopsy in a six year old girl who died of subacute bacterial endocarditis in spite of massive antibiotic therapy. The adenoma was not felt clinically. (Table 1.)

Nine surgical cases had benign adenomas, all but one of them presenting as solitary non-toxic nodules. Two of these recurred after surgery, one due to enlargement of the pyramidal lobe, requiring a second operation. The other child has not been under the care of the Children's Hospital but is known to have a 1 cm. nodule at the right superior pole present without apparent change for three years in spite of thyroid medication.

There has been only one case of recurrent goiter after thyroidectomy for hyperthyroidism. This was a nine year old Negro girl with classic hyperthyroidism who had been treated at another hospital for over a year with thiouracil. She was transferred to the Children's Hospital and after preparation with iodine she was operated upon by an experienced surgeon. The surgeon said that this was the most vascular gland he had ever seen. Hemostasis was so difficult that the operation was terminated after right hemithyroidectomy and left superior pole ligation. After further iodine a second attempt was made sixteen days later but had to be abandoned because of bleeding. After further preparation the left lobe was finally removed one week later. Postoperatively she did very well, but six months later a nodule appeared beneath the keloid scar. This enlarged slowly over the next two years. Three

years after the original surgery a 3½ cm. nodule was removed from the remaining left lobe and isthmus, and proved to be an adenoma with cystic degeneration. Throughout this time her thyroid function was within normal limits by repeated laboratory tests.

when a recurrence in the left neck was proved by biopsy.

The second patient was a six and a half year old boy who had had a thyroglossal duct cyst removed in 1947. In 1950 a 2 cm. firm, fixed, apparently solitary nodule appeared in the

TABLE II
THYROID CARCINOMA IN CHILDREN

Patient	Sex and Age	History	Clinical	Pathology
G. L.	F, 7	4 mo.	Cervical nodes only; thyroid "not abnormal"	Papillary, follicular and alveolar carcinoma with node metastases
M. F.	M, 6½	2½ mo.	Solitary nodule clinically; multiple nodules at surgery	Papillary adenocarcinoma
L. O.	F, 11	4 mo.	Solitary nodule	Malignant adenoma (D. H. Sprong)
M. B.	F, 11	1 yr.	Multiple cervical nodes clinically; hard irregular right lower pole at surgery	Alveolar carcinoma; node metastases

Two additional patients with nodular goiter were seen but not operated upon at the Children's Hospital. One was a fourteen year old girl with hyperthyroidism and palpable nodules at the superior pole bilaterally. In spite of a strong recommendation she was taken home without surgery. Seven years later she was operated upon at another hospital in Los Angeles and the pathologic report indicated involuted hyperthyroidism. The nodules were not described in the reports available to us.

The other was a seven year old boy with cervical and axillary adenopathy, a hard, nodular lower pole of the left thyroid lobe and a generally "rubbery" feel of the left compared with the right. Thyroid surgery was not performed.

Three patients with cancer of the thyroid have been seen at the Children's Hospital. (Table II.) One was a seven year old girl first seen because of bilateral, hard, cervical glands but with no palpable abnormality of the thyroid gland. Cervical node biopsy revealed carcinoma of the thyroid. Virtually total thyroidectomy, bilateral radical neck dissection and tracheotomy were performed in one operation. She had bilateral involvement of the thyroid lobes and of the cervical glands. The tracheotomy closed soon after and she did well until September, 1954, one year later, thyroid gland. At surgery several other smaller nodules which had not been felt clinically were found in the right lobe. The original pathologic examination did not indicate malignancy, but in reviewing the slides an area of papillary adenocarcinoma was discovered. The boy was lost to follow-up for four years but has recently been found living in Fresno. He is reported to have many large hard nodes in the right cervical chain and a nodule in the thyroid gland, but no left neck masses. Because of religious beliefs the parents have thus far refused to permit further study or treatment.

The third patient was an eleven year old girl sent to Los Angeles for x-ray therapy after thyroidectomy and radical neck dissection in San Diego. She had had enlarged cervical nodes for many months prior to surgery and these had been considered inflammatory. Biopsy revealed cancer of the thyroid; thyroidectomy and radical neck dissection were performed in two procedures.

My associate, Dr. David Sprong, has had two cases of non-toxic nodular goiter in children. One patient was an eleven year old girl with a solitary nodule present for four months. At surgery this proved to be malignant adenoma. The other patient was a thirteen year old girl with a solitary nodule present only one month. At surgery this was found to be lightly

adherent to the ribbon muscles and a frozen section was read as thyroid malignancy, but the final sections showed that it was only fetal adenoma with no evidence of malignancy.

There have been no deaths from cancer of the thyroid in children in the last ten years in Los Angeles. In that same time 548 children have died from other forms of malignancy. Cancer of the thyroid is a rare cause of death in children.

A total of twenty proved cases of Hashimoto's thyroiditis have been seen in children, and of these six were nodular to palpation.[11,20,42] We have had two such patients. One was a seven year old girl with a history of thyroid enlargement for four years and a growing solitary nodule in the isthmus for six months. Radioiodine uptake studies preoperatively showed 9 per cent uptake in forty-eight hours. At exploration to rule out malignancy the pale, firm, globular mass proved to be Hashimoto's thyroiditis. She has been well for one year since surgery.

The second patient was a ten year old girl with a history of definite pressure symptoms for seven months. Several nodules were found by clinical examination and at surgery. The original pathologic report showed hyperplasia with adenomatous changes, but in review of this the diagnosis of Hashimoto's thyroiditis has been made. A marked hypothyroid condition developed with a cholesterol level as high as 445 mg. per cent but is now fairly well controlled on 3 gr. of thyroid daily.

The University of California group found Hashimoto's thyroiditis in 39 per cent of hyperthyroid patients after radioactive iodine treatment compared to only 3 per cent after conventional preparation for surgery.[5] They believe that Hashimoto's thyroiditis is one result of the use of radioactive iodine. One of their cases of Hashimoto's thyroiditis was a five year old girl, possibly the youngest patient ever treated with radioactive iodine for hyperthyroidism.

There is a significant relationship between Hashimoto's thyroiditis and malignant thyroid neoplasms. In the California series malignant neoplasms occurred four times as often in those thyroids with some degree of Hashimoto's thyroiditis as in glands which did not contain it.[20]

As an additional sidelight, in the Memorial Hospital series nine of twenty-eight children with cancer of the thyroid had had irradiation of the thymus some time before the nineteenth month of life.[8] Freedberg's case[10] was a twelve year old boy with cancer of the thyroid who had had irradiation of the thymus at the age of eleven weeks.

These reports suggest that it is potentially dangerous to subject a child to internal or external irradiation of the neck for benign disease.

About 200 cases of carcinoma of the thyroid in children have been reported, as summarized by Winship[46] from American sources. The distribution of these cases is hard to explain. Winship has said, "It cannot be explained why no cases have been seen in Los Angeles but 11 cases have been reported from less populous San Francisco, nor why in Philadelphia only one case has been seen although 6 have occurred in Washington, D. C., a city in the same general area. Eighteen have been reported from the Mayo Clinic and yet Noble at the Ancker Hospital, Minneapolis, also in the heart of the goiter belt, has seen no thyroid carcinoma in children in 40,000 surgical specimens and none in 11,000 autopsies. Crile has had only 3 cases at the Cleveland Clinic in more than 25,000 thyroidectomies and Crotti, of Columbus, Ohio has seen only one thyroid carcinoma in a child although he has personally performed more than 12,000 thyroidectomies. Chesky states that there has not been a single thyroid carcinoma in a child during the last 33 years at the Hertzler Clinic in Kansas and Angrist knows of no recorded cases at Queens' Hospital, New York. Butt states that the surgical records of the Los Angeles County Hospital since 1918 show no children with thyroid carcinoma and none was found in 40,000 autopsies." A more recent letter from Dr. Butt, dated February, 1955, indicates that there have been three cases of carcinoma of the thyroid in children in the surgical material at the Los Angeles County General Hospital but no autopsy cases. All three surgical cases were seen within the last eight years.

The conclusion has been repeatedly drawn from the literature that the incidence of carcinoma in non-toxic nodular goiters in children is between 30 and 40 per cent. We suggest that it is misleading to offer this conclusion to our pediatric colleagues for two reasons:

First, we believe that in presenting statistics

cancers of the thyroid which can be diagnosed clinically should be separated from nodular goiters which clinically could be either benign or malignant.

Second, and perhaps more important, carcinoma of the thyroid in children is very likely to present with cervical node metastases and *no palpable abnormality of the thyroid.* Half of our cases and one-third of the Memorial Hospital series presented in that way. There are at least twenty examples in the literature.[1–3,8,12,15,18,43,46]

SUMMARY

The treatment of choice for congenital goiter causing respiratory obstruction is thyroidectomy. This operation should be as radical in children as in adults. X-ray therapy to the thymus gland may be hazardous and internal irradiation with radioactive iodine is accompanied by a striking increase in the incidence of Hashimoto's thyroiditis. There is a statistically significant correlation of Hashimoto's thyroiditis with malignant thyroid neoplasms. Carcinoma of the thyroid in children is predominantly papillary and very often is first seen with cervical node metastases but no palpable abnormality of the thyroid. It seems likely that if cases of cancer of the thyroid which can be diagnosed clinically and those cases which have no palpable abnormality of the thyroid gland were considered separately, the incidence of cancer in thyroid nodules in children would be much less than 30 to 40 per cent. In our experience it has been less than half of that.

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DISCUSSION OF PAPERS BY WARD, AND NORRIS AND POLLOCK

Dean B. Seabrook (Portland, Ore.): I have listened to the paper by Drs. Norris and Pollock with great interest. It has awakened again the fear of nodular goiters in children which was instilled in me by my beloved preceptor, the late Dr. T. M. Joyce; although, so far as I can remember, neither he nor I ever had a case of carcinoma of the thyroid in a child, i.e., below the age of fifteen. It is obvious from this report and many others, including articles by such authorities as Ward, Winship and Pemberton, however, that they are not extremely uncommon.

In 1951 Winship reported 198 cases from this country and in 1953, forty-two cases from Western Europe.

Pemberton states in his recent monograph that 2 to 3 per cent of all carcinomas of the thyroid occur in children fourteen years of age or less. It seems strange indeed that in the Portland Clinic series during the eight and a half years I was Dr. Joyce's assistant (during which time I personally examined and followed to surgery approximately 3,500 cases of goiter) no childhood carcinoma should be encountered, although 1 per cent of all cases coming to surgery were cancerous.

During the last ten years of my practice I have seen a relatively large number of cancers of the thyroid but none at all below the age of fifteen. All children with nodular goiters have been advised to have immediate surgery, however, because of my early training. The recent literature seems to substantiate this view. Pemberton, for example, stated in 1954 that one-third of all nodular goiters in children are cancerous. Within the last few years at the tumor clinic at St. Vincent's Hospital we have seen two children with carcinoma of the thyroid, one four and a half years of age, the other twelve. They were patients of Dr. Wilmot Foster and Dr. Arch Diack, both of whom consulted me, among others, regarding treatment. I advised radical surgery, much as advocated by the authors of this paper. Dr. Tom Fox has informed me of another child, ten years of age, with carcinoma of the thyroid who appeared at the tumor clinic at the University of Oregon Medical School. All of these children have undergone surgery and all are alive from one to five years after diagnosis; one, however, has persistent disease despite total thyroidectomy and staged bilateral radical neck dissection. None of these cases are included in Winship's 1951 report.

My associate, Dr. Nat Wilson, and I have been attempting to confirm something suggested by Ward in 1944, namely, that carcinoma of the thyroid preoperatively diagnosed has an ominous prognosis. Only 20 per cent in Ward's series lived five years when diagnosed prior to surgery. We have collected 103 cases from St. Vincent's, Providence, and the County Hospitals in Portland and are attempting to find out how long these patients will live. So far, after five years, twenty-seven are dead; 20 per cent from carcinoma. It is astounding to me that in this series of 103 cases we should find three in children. Even more surprising is the report by Harper et al. in the January issue of *Northwest Medicine*. In a series of 371 nodular goiters operated upon at the Deaconess Hospital in Spokane, only seven patients were below the age of twenty-one and all seven had carcinoma.

The recent article by Kriss in the January 8th issue of the *J. A. M. A.* on the formation of nodular goiters in children treated for anemia by a preparation containing cobalt awakens us again to the possibilities of carcinogens. Thiocyanates and

thiourea derivatives have yielded significant information on the relation of hyperplasia to neoplasia. Broders and Parkhill point out that in thiocyanate goiter cellular hypertrophy produces a picture comparable microscopically to the goiter of Graves' disease, whereas thiouracil-induced goiters show a cellular hyperplasia with mitosis suggestive of carcinoma. Both benign and malignant tumors in the thyroid of rats have been produced by Bielschowsky by feeding them with the carcinogenic agent 2-acetyl amino fluorine and allyl thiourea. Carcinomas and adenomas have been produced in rats by Money and Rawson by prolonged treatment with thiouracil alone.

A few years ago Dr. Arthur Curtis in Portland expressed the fear that thiouracil might be carcinogenic, and Foote found exaggerated hyperplasia with mitoses at times in glands of patients treated with thiouracil.

All this plus Norris' and Pollock's observations on the use of I-131 and the development of Hashimoto's thyroiditis and the relationship of this latter condition to carcinoma seem to emphasize several important considerations, namely, nodular goiters in children should come to surgery. All other methods of treatment are ineffectual and at times dangerous. Furthermore, treatment of toxic goiter with thiourea derivatives should be reconsidered in the light of these experiments. Certainly cobalt preparation for children should be avoided pending further investigation of its possible toxic effects.

I do not want to enter into the controversy concerning radical surgery for carcinoma of the thyroid, but having seen a number of cases limited to the lymphatic chain which has extended into the mediastinum I wonder how local radical excision could help if a cure is contemplated. It is true, of course, that many patients with cancer of the thyroid are alive many years after limited resections. It is also true, I believe, that the failures I see are more often than not the result of too little, not too much, surgery.

One other point in regard to classifications: many of the glands I have studied are mixed in character. One finds papillary formation, carcinoma simplex and adenocarcinoma in adenoma—all in different fields of the same gland. Unless a pure type is found, certainly radical surgery is justified if the case is operable and the surgeon believes cure is possible.

The many facets a paper like this brings out cannot possibly be considered in the time allotted and I have touched upon only a few. I wish again to state how stimulating this paper has been to me and to thank the authors for the privilege of discussing it.

EDWIN G. CLAUSEN (Oakland, Calif.): Dr. Ward has had a very keen interest in carcinoma of the thyroid for many years. Fortunately, carcinoma of the thyroid in children is uncommon, although one may not get that impression after talking with Dr. Ward. At The Children's Hospital of the East Bay there has been one case of carcinoma of the thyroid in 92,109 admissions in the past twenty-five years. This patient, a nine year old girl, had a papillary type which seemed to be relatively inactive in that small nodes in the neck were noted for three to four years before being biopsied. Positive nodes were noted on each side of the neck. No osseous or pulmonary metastasis had been noted. There was essentially no radioactive iodine uptake. Because of severe asthma, stage operations were performed. Total thyroidectomy revealed papillary carcinoma of the right superior pole. This was not palpable preoperatively. Following total thyroidectomy the patient was placed on 1½ gr. of thyroid and the family refused any further treatment. However, three years later, because the glands in the right side of the neck started to show some signs of activity, right radical neck dissection was performed which revealed numerous papillary metastatic nodes. The child has now been under our observation for five years and no therapy other than biopsy has been given to the glands in the left side of the neck which have remained essentially the same since they were first noted.

One would gather from the cases reported that papillary adenocarcinomas consist of about 50 per cent of the total. Although the other types are likewise relatively slow-growing, the prognosis is not as good. Dr. Ward has illustrated the use of all types of therapy. I believe he will agree with me that surgery should be the primary approach and that radioactive iodine and x-ray therapy be held in reserve for the management of lesions beyond the surgical field. Radioactive iodine is most ideal, but unfortunately it is of relatively little value since so few carcinomas will take the iodine in sufficient quantity. X-ray therapy may be very effective, but not curative. The use of substitution therapy after total thyroidectomy has been called to our attention.

ALSON KILGORE (San Francisco, Calif.): May I ask (and I am sure Dr. Ward will remember the patient who prompts the question) whether in his experience it has appeared that pregnancy may precipitate recurrence of previously operated cancer of the thyroid?

LEON GOLDMAN (San Francisco, Calif.): Dr. Ward has pointed out that carcinoma of the thyroid is often associated with great longevity which emphasizes the fact that few deaths occur during childhood from this disease. Carcinoma of the thyroid is a slow-growing, sluggish tumor which

may remain unchanged for many years, and there is evidence in many cases that it starts as carcinoma, presenting the nodule recognized clinically.

Vital statistics on public health records probably do not give us any clue as to the prevalence of this malignancy, but hospital and clinic reports indicate an increasing incidence. We may question our methods of recording the incidence of carcinoma in nodular goiter, a point which Dr. Pollock brought out in his report. The practice of adding together the cases of nodular goiter and the cases of carcinoma of the thyroid in a series and then concluding that the number of malignant cases constitutes a certain percentage of all nodular goiters is not logical. Some goiters are not palpable, but the diagnosis is made from metastasis; in other cases the palpable mass is the carcinoma and should not be considered as "nodular goiter." Dr. Pollock has made a definite contribution to this aspect of the problem.

ROBERTSON WARD (closing): One question asked is of great importance, that is, the administration of thyroid to youngsters with malignant goiter. It may be that this depresses the production of thyroid-stimulating hormone by the pituitary. In any case it is my habit to give tolerance doses of thyroid substance and in a number of cases I feel certain it has decreased the stimulus to recurrence and growth of malignant tissue. Dr. Clausen has reported one case which tends to bear out these findings.

When one discusses the pathology of malignant goiter, especially the papillary variety, there is always room for an argument. I have submitted papillary tumors to pathologists many times and asked them to tell me which growth was benign and which malignant. As often as not the one which has run a benign course for ten to fifteen postoperative years will be considered more malignant than the one to which the patient has succumbed in rapid order from recurrence and metastases. Certainly people can live with the disease for many years in apparent good health and in some a follow-up of twenty years is necessary to actually prove that the tumor is malignant. In children, in order for the growth to have a good prognosis, it is not necessary for it to be of the papillary variety as demonstrated by two of the cases reported. Evidently the prognosis is more dependent upon the individual's biologic resistance to carcinoma than it is to the presence or absence of the papillary pattern.

One of the questions asked was about bilateral neck dissections performed during one operation. I have never performed this in an adult but certainly there were no immediate or long-range undesirable effects in the little girl on whom it was done in this series. I would not hesitate to do the bilateral dissection at one sitting in any case in which bilateral metastases were demonstrable. There is probably a bigger safety factor in young people than in adults.

Someone has asked about a mixed pathologic picture. Practically all carcinomas of the thyroid are of a mixed pattern. I classify mine according to the predominant pattern and believe that this has some bearing on the prognosis.

Regarding the possibility that these youngsters are born with malignant goiters, I do not think this can be disproved. One of our patients was so well developed at the age of three and a half years that one could almost assume malignant goiter was present at the time of birth.

Time seems to mean little in carcinoma of the thyroid, especially in youngsters. The patients live so long with cancer of the thyroid that one has to have a different attitude about it than for cancers of other organs. It sometimes takes years for a case in which there was doubt at operation to prove itself either malignant or benign by the patient's death from metastases or unrelated causes.

In answer to Dr. Kilgore's question about the relation of pregnancy to cancer of the thyroid, I would say that in my experience and from what I have gained from perusal of the literature there is no such relation as we often see in cancer of the breast. Certainly there is one sex link regarding cancer of the thyroid which should be pointed out, and that is the greater likelihood of a nodular goiter being malignant in males than in females when cyclic changes in the gland associated with pregnancy and menstruation tend to make involutionary nodules very common in older women. In other words, the physiologic changes of the thyroid gland which are sex-linked in women do not take place in men and there is therefore no influence other than neoplasia which encourages nodular goiter to develop in men.

WILLIAM J. NORRIS (closing): A tremendous amount of work and study has been given by Dr. Pollock in the preparation of this paper. The outstanding point which in my opinion cannot be overemphasized is that an undiagnosed, persistent nodule in a child's neck should be biopsied. Our pediatrician friends should be made aware of this fact.

It has been a pleasure to participate in the preparation of this paper.

Electrolyte Imbalance in Postoperative Obstructions at the Gastrojejunal Stoma

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THE occasional failure of the stomach to function efficiently following gastrectomy has concerned general surgeons since Billroth's first successful resection in 1882.[1] During the ensuing period many concepts have been advanced to explain this sequel to resection, only to be refuted later by more accurate studies or improved research.

An obstruction of major concern follows a variety of surgical procedures for the treatment of peptic ulcers and gastric tumors, and occurs in about 5 per cent of gastrectomy patients.[2] Temporary manifestations which can be regulated by aspiration or restriction of oral intake are more common. This complication is rarely due solely to one actual defect. When reoperation is performed for revision of a possible extrinsic obstruction, a normal-appearing stoma is generally found. The failure to function properly is now believed to be due to gastric atony or neuromuscular imbalance.[3]

The belief that failure is caused by some nutritional inadequacy due to the prolonged dietary deficit imposed upon the patient because of pain or vomiting has led students to ponder a relation in this impaired healing to a depressed serum protein level. In 1948 Chauncey and Gray disproved this by their observation that gastric retention occurred in association with a serum protein level that was within normal limits and that the amount of retention did not parallel the serum protein concentrations. In fact at times the edema of hypoproteinemia was noticed postoperatively at the time gastric retention finally disappeared.[4,5]

Chlumskij, one of the early associates of the Polish surgeon Mikulicz,[6,7] theorized at the beginning of the century that some technical error in the nature of spur formation at the anastomotic site was the responsible agent. On this basis many studies have been made on the type of anastomosis, its position in relation to the colon, length of jejunal loop and questionable valve action to the gastric mucous membrane. As recently as December, 1953, at the Western Surgical Association meeting in Chicago, Prohaska proposed that this efferent stoma dysfunction is caused by an atonic gastric pouch which fails to function because the efferent outlet is placed above the afferent inlet.[8]

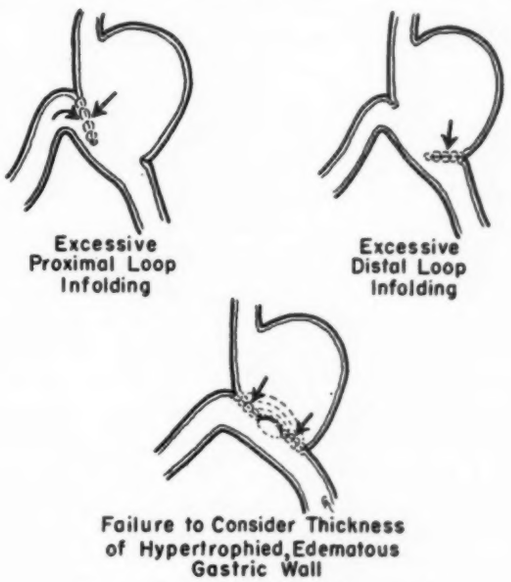

FIG. 1. Cause of obstructive spurs.

Although this thought is not accepted without reservation, the increased frequency of the complication in patients operated upon by junior fellows of surgery or by those who perform this type of surgery less often than experienced gastric surgeons lends weight to the possibility that some minor technical or mechanical fault initiates the retention or ileus.[30] There is little difference as to type of operation. In this study several procedures are considered partially responsible because they appear most common in the obstructed group. Probably because of the fear of leak at the anastomosis a novice surgeon attempts to reinforce the angles

by additional or too deeply placed sutures which can give origin to spur formations. (Fig. 1.) In the patient who has had prolonged pyloric obstruction without adequate preoperative decompression and proper electrolyte replacement, an obstructive cuff will on occasion develop due to the edema within the thickened gastric wall. The two most common surgical errors observed in posterior anastomoses have to do with the position of the rent in the mesocolon. (Fig. 2.) In high resections which have later obstructed, the mesocolon has been sutured too closely about the anastomosis; and as healing has taken place, the tension of the colon has produced a stenosing effect about the suture line. In the sthenic type of build the rent in the mesocolon has at times been placed too far to the right; and when the stomach has been returned to its natural position, the anastomosis is angled or distorted causing obstruction. (Fig. 3.)

During the past decade an improved knowledge of fluid balance and electrolytes has aided the student of this problem by directing his attention in solving this to studies of the sodium, potassium and chloride imbalances which occur with this complication. Although a potassium depression has been thought to be a factor in association with the alkalosis and hypochloremia, the biochemical determinations of the plasma potassium have not shown consistently low levels. It has been recognized, however, for some time that the clinical improvement in these patients was sudden and striking after adequate amounts of potassium had been administered.[9]

The frequency with which a stoppage occurs at the anastomosis between the stomach and jejunum and its rarity in enteroenterostomies and colocolostomies is related to the disproportionate losses of potassium and chloride that develop in selected patients following gastrectomy.[12] This disruption effects changes at the intracellular level. The upper secretory intestine normally requires more abundant reserves of these ions because of its greater exchange demands for them over those of the lower absorptive intestine. Acute gastric dilatation

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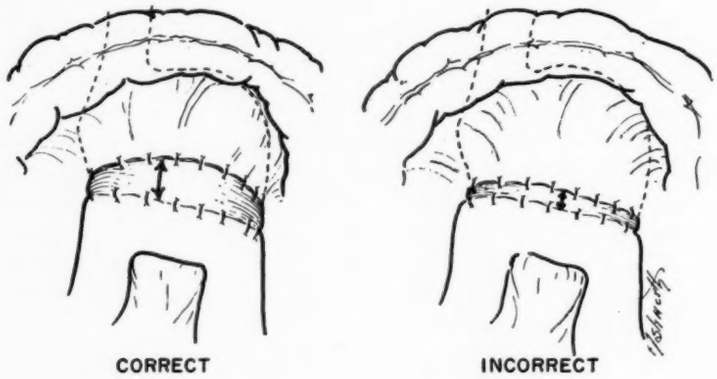

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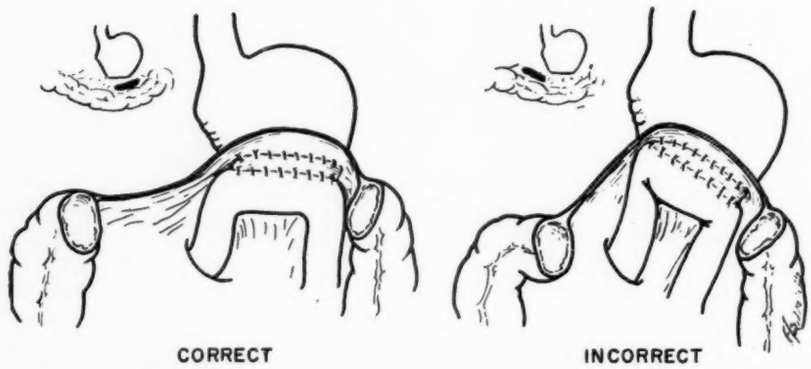

FIG. 2. Obstruction due to improperly placed mesocolon.

FIG. 3. Obstruction due to improperly placed rent in mesocolon.

and proximal ileus are related to this event in that they, too, share an imbalance in potassium and chloride losses which plays the significant part in the pathogenesis of the neuromuscular paralysis in all three.[10,26] Logan has attributed the gastric ileus which on occasion follows gastroenterostomy, to a potassium depletion.[3] MacPhee[11] has elaborated on this problem, and Moore[15–18] has applied the principle of isotope dilution by studies on measurements of the artificially produced potassium K-42 and sodium Na-24 isotopes.

An intracellular potassium depletion is common in postgastrectomy patients and produces a disorder of muscular contraction in the intestine similar to that which is seen experimentally in animals.[9] This is in contrast to the familiar neuromuscular induction disorder which is seen in the less frequently occurring extracellular deficiencies and is expressed by changes in the electrocardiogram. This body cell K depletion develops in any patient who has been losing extrarenal body fluids through aspiration or vomiting for two or three days and is demonstrated by measuring the total body potassium by K-42 dilution in contrast to a plasma K depletion which is determined by the flame photometer.

It is unwise to wait for biochemical evidence of serum potassium depletion before making the diagnosis. With adequate renal function, a deficiency must be expected in all patients who have undergone gastric resection if oral feeding is not possible by the fourth day, if oral intake has been restricted before operation, or if these losses have been augmented by vomiting after surgery. Dehydration and the injudicious use of saline infusions are contributing agents in causing the shift of potassium.

K-42 is the best known of the artificially produced potassium isotopes.[13–15] It emits two beta and one gamma particle and is produced by the cyclotron or at the atomic pile from a natural-occurring isotope K-41. For these studies it is received in the carbonate form, neutralized, diluted and injected intravenously into the patient. It has a half life of 12.4 hours and is measured by a variety of methods to determine the intracellular levels. Generally this is done by counting assays of blood and urine with the Geiger-Müller tube and conventional scaler.

The gradient of extra- to intracellular potassium across the cell membrane is referred to as a biologic or dilutional readjustment as contrasted with an osmotic equilibrium.[10] The exchange of these ions requires hours or days depending upon the tissue involved, and for this reason the cell membrane is called a slow one to potassium. Equilibrium is attained throughout the body in radioisotope experiments in about twenty hours. This delay partially explains why the plasma potassium levels are unreliable indices of the state of intracellular affairs.

Acetylcholine formation and the process of nerve conduction are dependent upon a normal intracellular potassium gradient.[10] This choline compound formation is essential to the tone and movement of the intestine and its most active area is in the upper intestine. Its liberation at the ganglionic synapse is apparently effected only in the presence of an adequate concentration of potassium ions with phosphorylated sugars which provide the energy.[21,22,28]

A normal intracellular potassium demand is approximately thirty-one times greater than the extracellular while the intracellular sodium requirement is twenty times less than that of the plasma. These gradients appear to be reciprocal. Body total potassium is unevenly distributed between the spaces[28] and is transferred from the cells in the postoperative phase even more rapidly than it is excreted in the urine. The kidney seems unable to conserve it. This continued excretion in the urine in a patient already in imbalance is an important factor in producing potassium deficiency[23] following major gastric operations and is greater in proportion than the nitrogen loss.[24] This accounts for the clinical findings of potassium deficiency in the presence of a normal serum concentration of the ion. Increased adrenocortical activity may be the cause of potassium withdrawal from the cells.

If a plasma potassium concentration is lower than 4 mEq./L., if the acidity outside the cell is increased or in conditions of alkalosis due to excessive $NaHCO_3$ in the plasma, potassium leaves the cells for the extracellular fluid compartment. This transfer is later balanced by a shift of sodium into the cells. When potassium leaves the cell, it is replaced at first by sodium weight for weight but later by excessive sodium which interferes with many enzymatic reactions in the cell;[25] and this cellular sodium demand may prevent the excretion of urinary

sodium. If the parenteral intake is limited to sodium chloride, there is the possibility that the sodium excess within the cell will produce an abnormal electrolyte pattern.[29] Extensive tissue injury following complicated gastric surgery, prolonged shock due to hemorrhage, anoxia and unrecognized or untreated cellular dehydration will also cause transfer of potassium.

To correct this condition the potassium deficit must be made up by the administration of potassium orally or parenterally. To force the potassium from the plasma and interstitial fluids into the cells it appears that glucose and insulin must be available. These cause phosphoric acid to be converted into more acidic sugar phosphate esters within the tissue cells, and potassium then moves into the cells to neutralize this increased acidity. Glycogen is then deposited into the cells by the movement of potassium.

During the first four days after surgery the adrenal cortical hormone produces potassium loss and sodium retention. These changes and the associated alkalosis result in varying degrees of ileus. Following this period gastric aspiration or vomiting will cause loss of more chloride and potassium ions as the gastric contents contain two to three times as much potassium as an equivalent amount in the serum.[27] Chloride waste is in excess of sodium due to free HCl in gastric secretion. Even though gastric juice is without titrable free HCl, large amounts of the ion are lost as sodium and potassium chloride. This accounts for the increasing alkalosis. Potassium is present in much the same concentration in the gastric secretions of patients with either ulcer or carcinoma.[20] These losses in the second week are manifested clinically by gastric dilatation, dehydration and alkalosis provided there is no potassium replacement after the first four or five days.

Part of the intracellular potassium is bound to protein and part is exchangeable. The bound fraction 2.8 mEq. K/gm. N moves with the cell nitrogen and is excreted as protein is broken down. It is retained in growth and healing. The convertible fraction moves with the cell water as metabolic processes change and manifests its maximum lability in the alimentary canal involuntary muscle.[10]

In the secretory intestine the absorption and secretion of water and electrolytes is five times greater than in the absorptive intestine. Unless blood changes occur, the mineral composition of the digestive juices follows a definite pattern. The modified juice always reflects the secretory potential of the tissue as shown by the high chloride-secreting qualities of the oxyntic cells which continue despite gastric hypersecretion which is associated with alkalosis due to losses of K and chloride ions. These two are essential to the digestive juices of the proximal intestine. The quantity of these involved is very substantial when compared with the daily intake or with the plasma levels. The lower requirements of these in the distal intestine determines its earlier recovery in deficient states and emphasizes the difficulty of restoring the function of the secretory intestine.

Magnesium[19] resembles potassium in body fluid distribution in its intracellular importance. Its cellular concentration averages about 28 mEq./L. and it apparently takes part in energy transfers involving the enzyme, adrenosine triphosphate. The normal serum level is low, averaging 2 mEq./L. Like potassium, the serum levels of magnesium do not necessarily indicate the intracellular or total body levels. Reports in the literature of low serum magnesium levels appear to be unassociated with any known symptom complex.

Peptic ulcer and gastric tumor patients who have been on restricted diets or who have had prolonged parenteral therapy may show a deficiency of magnesium. The main source of supply of this ion is in the chlorophyll of green vegetables and these are frequently not included in such diets. The renal excretion of magnesium follows the pattern of sodium in conservation and for this reason depletion occurs less readily than with potassium. Twelve to seventy hours are needed to correct intracellular defects when treated by magnesium sulfate.

X-rays and brief summaries of four obstructed postgastrectomy cases are submitted to show the typical x-ray findings of the neuromuscular imbalance which occurs at the stoma, and the associated chloride and alkali reserve findings. All of these patients recovered after adequate intake of the needed electrolytes. One patient (Case No. 109838, Rupert) was operated upon after the method of Allen,[2] and both draining and feeding jejunostomy tubes were inserted.

X-rays (Fig. 4) of Case No. 118221 (S. V. H.)

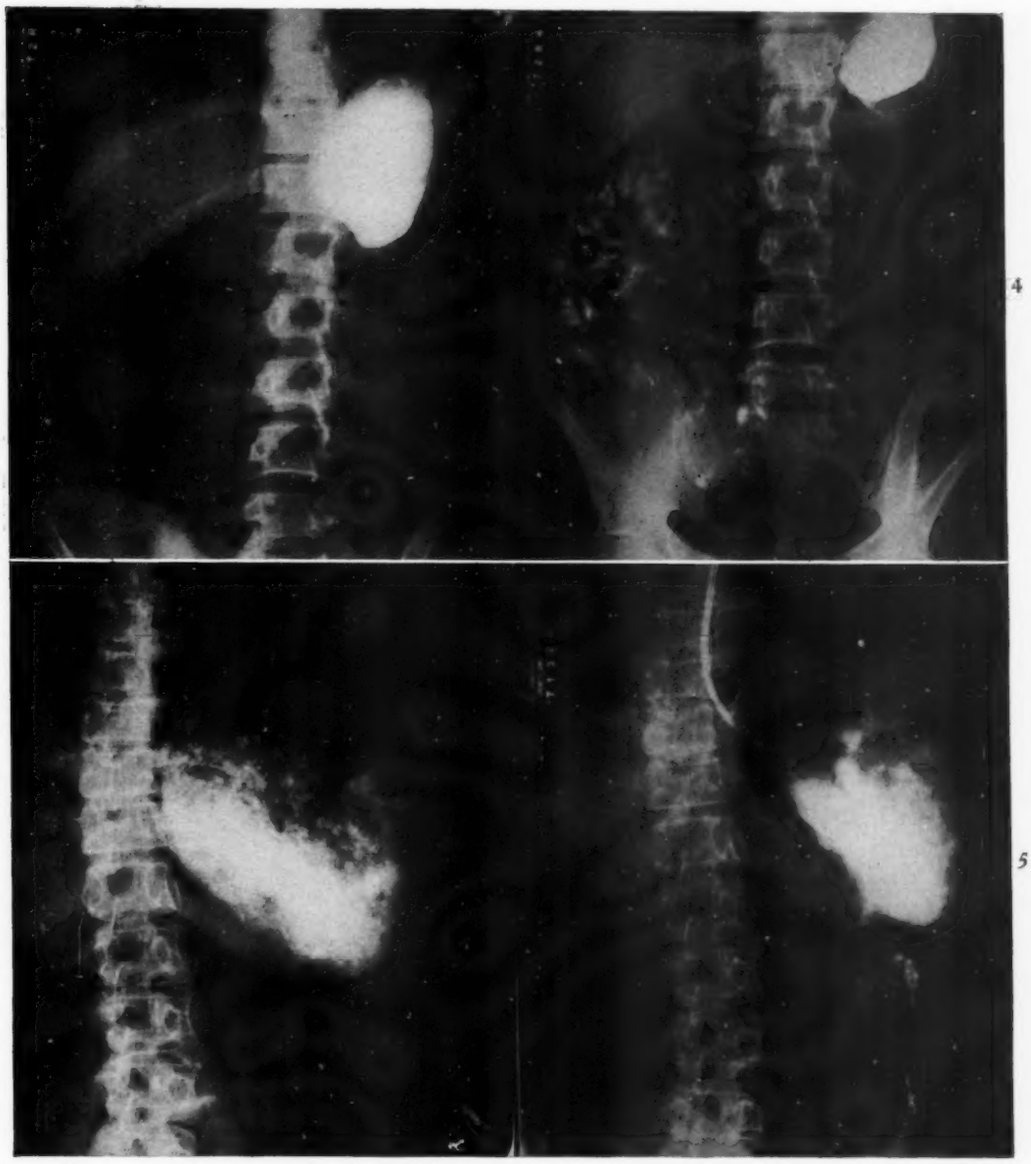

FIG. 4. Case 118221. Gastric ulcer; obstructed fifteen days postoperatively. Chloride as NaCl 350 mg. per cent, 60 mEq./L. plasma; alkali reserve 110 volumes per cent (carbon dioxide combining power), 49 mEq. HCO_3/L.; urea 53 mg./100 cc. Both x-rays were taken on January 4, 1954 (three-hour difference).

FIG. 5. Case 56905. Duodenal ulcer; obstructed sixteen days postoperatively. Chloride as NaCl 462 mg. per cent, 79 mEq./L.; alkali reserve 97 volumes per cent, 43 mEq./L. The x-ray on the left was taken at onset of obstruction, September 20, 1952. The x-ray on the right was taken five days later, September 25, 1952.

demonstrate a stomal obstruction which developed fifteen days following surgery for a benign gastric ulcer. The contrast shadow of thorium dioxide in the stomach reveals the neuromuscular imbalance at the stoma, and the biochemical evidence of the associated alkalosis and hypochloremia is expressed in the legend. The film to the right shows a trickle of the contrast medium passing into the efferent loop of the jejunum the following day. Complete drainage was established after a normal electrolyte pattern was attained, which followed after the sodium, potassium and chloride imbalances had been corrected. Potassium chloride (3.0 gm. KCl/L.) was administered with 5 per cent glucose intravenously.

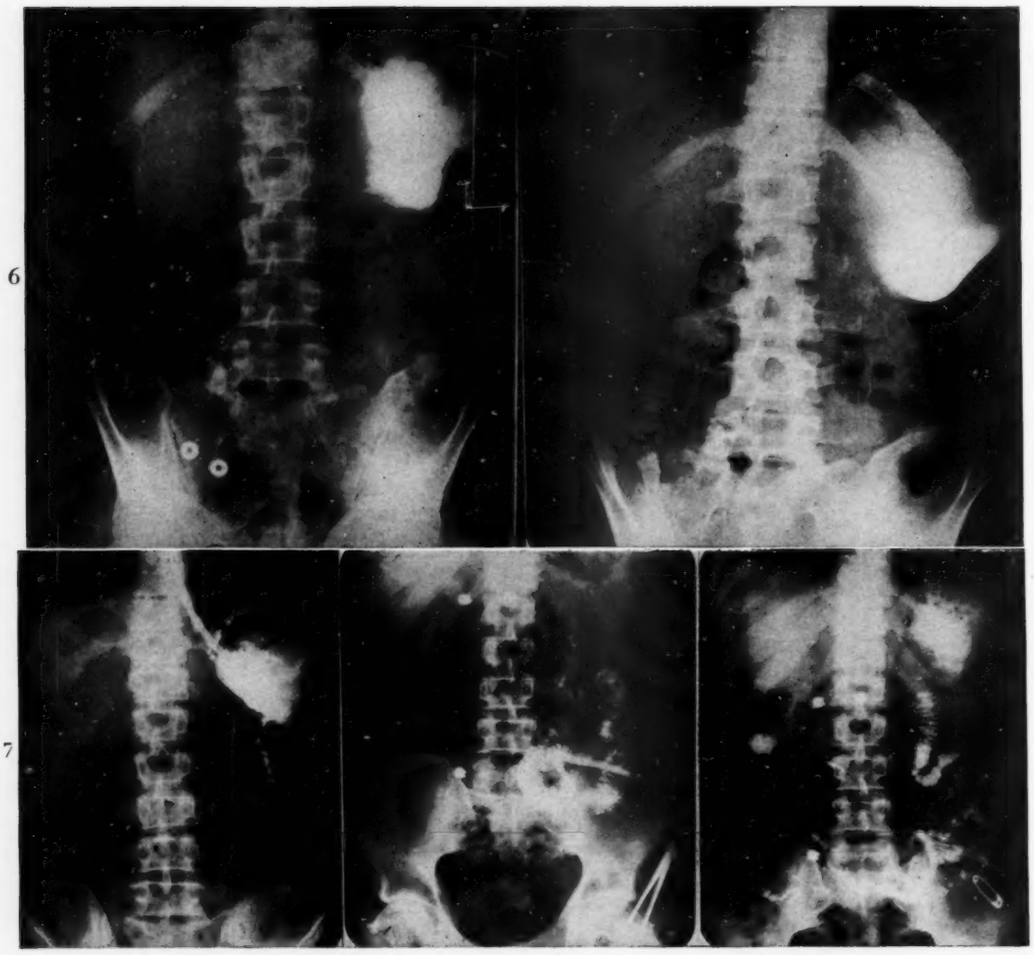

FIG. 6. Case 80489. Gastric ulcer; obstructed twelve days postoperatively. Chloride as NaCl 581 mg. per cent, 100 mEq./L.; alkali reserve 82 volumes per cent, 36 mEq./L.; urea 5 mg./100 cc.; protein 6.8 mg./100 cc., 16.5 mEq./protein/L. plasma. The x-ray on the left was taken April 13, 1953. The x-ray on the right is of a twenty-four-hour retention April 14, 1953. The electrolyte findings were those recorded eight days after the obstruction was diagnosed and treated.

FIG. 7. Case 109838. Duodenal ulcer; obstructed seventeen days postoperatively. Chloride as NaCl 429 mg. per cent, 73 mEq./L.; alkali reserve 125 volumes per cent, 56 mEq./L.; urea 9 mg./100 cc.

Figure 5 illustrates Case No. 56905, a middle-aged woman who had obstruction sixteen days after gastric resection had been performed for an obstructing duodenal ulcer. Both x-ray films show the characteristic retention or atony after a swallow of thorium dioxide. The x-ray to the right shows the gradual leakage into the jejunum. The disproportionate chloride and potassium losses and the high alkali reserve were corrected at the same time drainage was established.

X-rays (Fig. 6) of Case No. 80489 are similar. The obstruction occurred at the usual time, but eight days of carefully calculated and administered fluid replacements were required to correct the ileus and alkalosis, to overcome dehydration and to re-establish an adequate serum chloride level and a normal potassium gradient.

Figure 7 shows an umbrathor study on Case No. 109838. In the first film the characteristic stomal obstruction and corresponding electrolyte imbalance are seen. The middle photograph is a test for patency of the distal loop—a jejunostomy feeding tube after the method of Allen had been previously placed. Thorium dioxide was introduced into this tube to prove the absence of distal obstruction. The film to

the right reveals normal gastric drainage after thorium dioxide had been given orally and the serum electrolyte levels were normal.

CONCLUSION

It should not be construed from this brief discussion of the electrolyte changes which occur in postgastrectomy obstructions that a normal convalescence after gastric resection should have meddlesome treatment. In the complicated case, however, after the fourth postoperative day if food or fluid by mouth is not tolerated, an energetic type of replacement therapy should be instituted.

SUMMARY

The failure of the stoma to function properly in the manner of gastric retention following gastrectomy in association with alkalosis and low plasma chloride is believed to be due to an abnormal loss of chloride and intracellular potassium.[31] The normal potassium gradient and its relation to cellular function in the upper secretory intestine is discussed. These facts have been confirmed by application of the principle of isotope dilution with studies using radioactive potassium and sodium.

Therapy should be directed in replacing potassium slowly (2.0 to 3.0 gm. KCl per L. of 5 per cent glucose) and avoiding the injudicious use of saline. Hypoproteinemia and ascorbic acid deficiency if present should be corrected.

Because of the increased frequency of this complication on the services of less experienced surgeons several technical surgical suggestions are offered as these were observed most commonly in the series studied.

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DISCUSSION

Clarence J. Berne (Los Angeles, Calif.): Dr. Roberts has very ably presented an extensive consideration of potassium deficiency as the cause of inertia of the gastric stump in patients with malfunctioning gastrojejunostomy. Surgeons are steadily learning more regarding the physiology of potassium and its great importance in gastrointestinal surgery. Certain fundamentals merit emphasis. The most basic is that renal excretion of potassium goes on day after day regardless of whether or not potassium is being acquired by the organism. In health, this is provided by the diet. Poor dietary intake for any reason will therefore throw the individual into potassium insufficiency. The kidney has no ability to conserve potassium. Renal excretion of potassium is increased by trauma and probably by alkalosis. If nasogastric suction is withdrawing significant volumes of secretion, an extrarenal loss of potassium is added. In ninety cases that we have studied, where nasogastric suction was being used and the parenteral fluids were potassium-free, lowered serum potassium levels developed on an average of 4:5 days if there had been a preceding week of poor food intake. These factors are inevitably operative in every case of gastrectomy. The average gastrectomy patient does well and corrects the deficiency when alimentation is begun. This is where the difficulty begins if stomal malfunction appears. The patient is already in potassium deficiency. For this reason we have, for the past five years, required of our residents that all gastrointestinal surgical patients be given 6 gm. of KCl daily. The exception is a patient with poor renal function. All this develops a principle in postoperative care. Potassium deficits are preventable and should be avoided. Their consequences are numerous and serious.

We believe in rapid correction of serious deficits. This is usually done by adding 3 gm. of KCl to the basic daily dose of 6 gm. As much as 20 to 30 gm. a day have been given.

A common pitfall in estimating deficiency is presented by the patient with malfunctioning gastrojejunostomy who is very apt to be dehydrated, with resultant elevation of the serum potassium to normal levels by hemoconcentration. If there is no reduction of plasma water, it has been our belief that acute potassium deficits are well identified by serum potassium determinations.

Dr. Roberts suggests that there was a syndrome of gastric stump inertia without intestinal paralysis, with normal serum potassium, with low intracellular K, in the cases he reports and that the use of potassium intravenously corrected the malfunction. This may be true. However, I cannot help but mention that fifteen years ago we saw such cases open up on the sixteenth or eighteenth day and potassium was, of course, not used. In order to clarify instances such as he reported it would be relatively easy to measure the daily urine output of potassium. If potassium therapy was then started, the renal excretion of potassium, in relation to the amount given intravenously, would give an index of the amount retained and give the clinician similar information to that obtained by research studies with K-42. This should be true even though the serum potassium levels were normal. Correlations with the clinical features could then provide a basis for further conclusions in regard to the relationship that he has so ably suggested. However, the most practical answer to the problem is to not permit the deficits to occur, thereby eliminating this pathophysiologic mechanism from consideration in the differential diagnosis of the cause of malfunctioning gastrojejunal stoma.

Joseph M. Roberts (closing): I would like to thank Dr. Berne for his comments and criticism for considering only a single electrolyte deficiency. I did not mention magnesium because of the time allotted to me. The studies with this electrolyte, however, would suggest that even though there may be a magnesium deficiency it probably has no effect on stomal retention.

Dr. Berne's suggestion that repeated urine potassium determinations may be a practical method of analysis in institutions not equipped for K-42 studies is an excellent one.

Peptic Ulcer in the Aged

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IN view of the present day indications for surgical intervention in peptic ulcer and the increasingly aggressive approach to the problem it was our desire to determine, under our working conditions, whether the same therapeutic concepts were valid in the older age groups.

Chronic ulcer of the duodenum and even of the stomach usually is considered as a disease of early or middle adult life. Yet clinicians are becoming increasingly aware of its occurrence in older people as well and it is claimed that the incidence is rising more rapidly than the per cent of increase in the number of elderly persons.[10] In any event, as life expectancy becomes progressively prolonged, the problem of dealing with peptic ulcers and their complications assumes greater importance. In the aged the disease may pass unrecognized because of lack of the typical ulcer symptom pattern, or proper treatment may be withheld because of the patient's age or a mistaken impression of inoperable malignancy. The literature abounds with reports of elderly patients dying from hemorrhage, perforation or even inanition due to peptic ulcer, many of whom might have been spared by correct diagnosis and proper therapy.[1,3,6,13]

There is no difference of opinion regarding the importance of correct diagnosis. Likewise, it is recognized that some complications of ulcer, such as hemorrhage, tend to become more frequent and more serious with advancing age.[13,14] There is, however, a lack of general agreement regarding therapy. Some authors believe that medical treatment is almost universally applicable.[11] Others, recognizing that old people tend to tolerate surgery well in this day and age, point out that many catastrophic complications and much suffering could be avoided by earlier elective surgical treatment and believe that the same indications for operation exist in the aged as in the remainder of the population.[1,3,4,6,14,17]

OBSERVATIONS

This study was confined to patients seventy years of age or older who were admitted to Sacred Heart Hospital with the diagnosis of gastric, duodenal or jejunal ulcer between January 1, 1951, and December 31, 1954. There were 135 such patients. In 111 the ulcer was the principal reason for hospitalization and its presence was confirmed by either x-ray examination, operation or autopsy. There were seventy-six men and thirty-five women in this group. Their ages ranged between seventy and eighty-eight years, the average being seventy-five years. In sixteen patients ulcer was present, but was an incidental finding and not the chief reason for hospitalization. In addition there were eight patients admitted because of massive hemorrhage, acute perforation or severe pain in whom the diagnosis was presumptive and not conclusive. Further consideration is limited to the first group of 111 patients. It should be stated at this point that the care of these patients fell to numerous different internists, surgeons and general practitioners who were members of the staff of Sacred Heart Hospital, an open-staff, general hospital in Spokane, Washington.

Location of Ulcer. The ulcer was located in the first portion of the duodenum in eighty-three patients (fifty-five men, twenty-eight women), in the stomach in twenty-one (fifteen men, six women). Five patients (all men) had both gastric and duodenal ulcers and two (one man, one woman) had jejunal ulcers. (Table 1.)

Predominant Symptom. Among the patients having duodenal ulcer, intractable distress was the chief reason for hospitalization in thirty-eight, hemorrhage in thirty-six, acute perforation in five and obstruction in four. In the gastric ulcer group eight patients were admitted because of distress, six because of acute perforation, five because of hemorrhage and

two because of obstruction. In eight of these gastric ulcer cases anxiety was felt because of the possibility of carcinoma. Among the patients having both gastric and duodenal ulcers four were admitted because of hemorrhage and one because of distress. Both of the patients having jejunal ulcers were admitted because of hemorrhage. It goes without saying that many of the patients who bled also had other ulcer symptoms. (Table II.)

TABLE I
DISTRIBUTION OF ULCERS

Location	Males	Females	Total
Duodenum	55	28	83
Stomach	15	6	21
Duodenum and stomach	5	0	5
Jejunum	1	1	2
Totals	76	35	111

TABLE II
PRINCIPAL SYMPTOMS

Location of Ulcer	Distress	Hemorrhage	Acute Perforation	Obstruction
Duodenum	38	36	5	4
Stomach	8	5	6	2
Duodenum and stomach	1	4	0	0
Jejunum	0	2	0	0
Totals	47	47	11	6

Previous Ulcer History. Most of the patients had suffered from intermittent distress before the present admission, but not infrequently the duration of symptoms was brief. Previous severe hemorrhages had been experienced by six patients with duodenal ulcer, one with gastric ulcer and one with jejunal ulcer. Closure of an acute perforation had been carried out in four patients with duodenal ulcer and one with gastric ulcer. One patient with an active duodenal ulcer had had gastroenterostomy performed and both patients with jejunal ulcer had been subjected to this operation. One patient with a large gastric ulcer had had previous gastric resection. Among the patients having both gastric and duodenal ulcers, one had had pyloroplasty and one a wedge resection. (Table III.)

TABLE III
PREVIOUS TROUBLE

Location of Ulcer	Hemorrhage	Closure of Acute Perforation	Other Operations
Duodenum	6	4	1
Stomach	1	1	1
Duodenum and stomach	0	0	2
Jejunum	1	0	2
Totals	8	5	6

TREATMENT

Medical management alone was afforded seventy patients (63.1 per cent). This was the conventional regimen of rest, diet, antacids and antispasmodics. Patients who bled were transfused. Feedings usually were continued, notwithstanding bleeding. Two patients with acute perforation were treated conservatively by intubation, suction, antibiotics and parenteral alimentation. (Table IV.)

TABLE IV
TYPE OF TREATMENT

Location of Ulcer	Medical	Surgical
Duodenum	62	21
Stomach	4	17
Duodenum and stomach	3	2
Jejunum	1	1
Totals	70 (63.1%)	41 (36.9%)

Surgical procedures were carried out on forty-one patients (36.9 per cent). This included twenty-one patients with duodenal ulcer (25.3 per cent), seventeen patients with gastric ulcer (81 per cent), two patients having both gastric and duodenal ulcer (40 per cent), and one patient with jejunal ulcer (50 per cent). Emergency operations were as follows: closure of acute perforation, nine (duodenal ulcer, four patients; gastric ulcer, five patients); resection for uncontrolled bleeding, four (duodenal ulcer, two patients; gastric ulcer, one

patient; jejunal ulcer, one patient); gastrotomy and ligation of bleeding duodenal ulcer, one patient. Elective operations were as follows: gastric resection, twenty-one (duodenal ulcer, ten patients; gastric ulcer, ten patients; gastric and duodenal ulcer, one patient); gastroenterostomy, five (duodenal ulcer, four patients; following wedge resection in patient having both gastric and duodenal ulcer, one); total gastrectomy, one (gastric ulcer). (Table v.)

TABLE V
OPERATIONS PERFORMED

Location of Ulcer	Gastric Resection	Gastro enterostomy	Closure of Perforation	Others
Duodenum	12	4	4	1
Stomach	11	0	5	1
Duodenum and stomach	1	1	0	0
Jejunum	1	0	0	0
Totals	25	5	9	2

Results of Treatment. Among the seventy patients undergoing medical treatment there were seven deaths (10 per cent). Five died from hemorrhage, one following acute perforation and one of acute myocardial infarction complicated by congestive heart failure. (Table vi.) The remainder of the patients left the hospital, temporarily at least, improved.

TABLE VI
CAUSE OF DEATH IN CASES TREATED MEDICALLY

	No.	
Hemorrhage	5	
Peritonitis	1	
Heart disease	1	
Total	7	(10 per cent mortality rate)

Among the forty-one patients operated on there were eight deaths (19.5 per cent). Four of these followed emergency procedures (28.6 per cent) and four followed elective procedures (14.8 per cent). (Table vii.) The remaining thirty-three patients ultimately left the hospital ostensibly improved. One-third of them had postoperative complications in varying degrees of severity. (Table viii.)

It is also of interest to consider results of treatment in relation to certain presenting symptoms. Among the forty-seven patients who bled there were eight deaths (17 per cent), five following medical treatment, two following emergency operation and one following elective operation. Among the eleven patients with acute perforation, there were three deaths (27.3 per cent), one after medical treatment, and two after emergency operation. Two of six patients with obstruction died following gastroenterostomy (33.3 per cent). Among patients treated for pain alone there were two deaths (4.3 per cent), one following medical treatment and one following operation. (Table ix.)

TABLE VII
FATAL POSTOPERATIVE COMPLICATIONS

	No.	
Hemorrhage from acute gastric ulcer	1	
Hemorrhage from ligated duodenal ulcer	1	
Peritonitis; coronary occlusion	1	
Evisceration; shock	1	
Evisceration; bronchopneumonia	2	
Evisceration; coronary occlusion	1	
Coronary occlusion	1	
Total	8	(19.5 per cent mortality rate)

TABLE VIII
NON-FATAL POSTOPERATIVE COMPLICATIONS

	No.	
Atelectasis; pneumonitis	1	
Severe upper respiratory infection	1	
Duodenal fistula; pneumonia	1	
Duodenal fistula	1	
Gastric retention	1	
Bile peritonitis	1	
Gastrointestinal bleeding; evisceration	1	
Esophagitis; malnutrition	1	
Ileus; wound infection	1	
Urinary retention	2	
Total	11	(33.3 per cent)

TABLE IX
DEATHS IN RELATION TO PRESENTING SYMPTOMS

Symptoms	No. of Patients	Deaths	Per cent
Distress	47	2	4.2
Hemorrhage	47	8	17.0
Acute perforation	11	3	27.3
Obstruction	6	2	33.3

COMMENTS

We were surprised at the large number of patients over seventy years of age who were

admitted to the hospital because of peptic ulceration during the four-year period of our study. The ratio of duodenal ulcer to gastric ulcer was 4 to 1. Reports in the literature range from 15 to 1,[14] at one extreme, to a reversal of the ratio at the other extreme.[13,15] The ratio of men to women was approximately 2 to 1 in contrast to the usual ratio of about 4 to 1 in younger people.[2] The most common cause for admission to the hospital among patients having gastric or duodenal ulcer was intractable distress. The most common complications were hemorrhage (among patients having duodenal ulcer) and perforation (among those with gastric ulcer). Obstruction occurred infrequently compared to the observations of others.[6,14] It is interesting to note that eight of the patients had had previous severe hemorrhage and eleven had undergone some type of operation. When one encounters patients who have had repeated admissions because of their ulcer and who finally expire from it or barely escape with their lives after a hazardous emergency operation, it is difficult to justify the conservatism which withheld an earlier definitive elective procedure. Certainly even elective surgery carries an increased risk in the aged with their often manifold infirmities. A list of the associated diseases from which our patients suffered would resemble the table of contents of a textbook of medicine. Yet a carefully planned and executed operative procedure, with good anesthesia and good pre- and postoperative care is not the debacle some would like to make it out to be. In our hospital, an open-staff institution where the operations are carried out by diverse individuals, the mortality rate was 14.8 per cent. This figure is comparable to that reported by others.[5,12] In contrast to this the mortality rate from complications regardless of type of treatment was 17 per cent for hemorrhage, 27.3 per cent for acute perforation, and 33.3 per cent for obstruction, and the mortality rate following emergency operation 28.6 per cent.

A study of the fifteen patients who died, regardless of whether medical or surgical treatment was carried out, is most illuminating. Among the eight patients who bled, one could not escape the feeling that two might have fared better with emergency operation, that two should have been operated on after previous milder hemorrhages and that emergency operation was carried out too late in one and inadequately in another. Two patients died following gastroenterostomy for obstruction. In one patient this was carried out after a previous inadequate operation; in the other ample indications for operation had long existed before the final debilitating illness. One patient with both gastric and duodenal ulcer, who had repeated admissions over a period of years, could have been saved much suffering and expense before his death from congestive heart failure. We could find no fault with the plan of management of the remaining six who died.

Analysis of the eight surgical deaths, included in those previously mentioned, simply emphasizes the fact that old people tolerate operation well, complications poorly. It is a shock to find that wound dehiscence was the precursor of death in four instances and suggests that greater precaution than ordinary conventional methods should be taken in closing these wounds initially. Postoperative complications among patients who survived were comparable to those seen in a younger age group, although in higher incidence. Only one patient survived wound disruption.

In general it would seem that the same indications exist for operation in the aged as in the younger patient. If this concept is neglected, unnecessary deaths occur. The unfortunate tendency to procrastinate and to regard surgery as a last resort is one reason mortality rates are as high as they are.

The average patient with duodenal ulcer is best treated medically; however, even in the aged if pain is not relieved, operation must be considered. We had an eighty-three year old man who threatened suicide because constant pain and self-imposed subsistence on goat's milk had rendered life intolerable.

Bleeding of any magnitude warrants serious consideration of operation. Most of the people who die from hemorrhage from peptic ulcer fall into the older age group.[13] If the bleeding can be stopped, surgery may be elective. If replacement of 1,500 ml. of blood in twenty-four hours does not keep ahead of blood loss, emergency operation is indicated. This was carried out successfully in two of our patients over eighty years of age. It seems unjustifiable to permit a patient to have one hemorrhage after another and finally bleed to death from what should be a remediable condition.

Acute perforation is best treated surgically.

Only thus can closure be surely effected and the location and character (benign or malignant) of the perforating ulcer accurately ascertained. Cases formerly regarded as hopeless because of shock can sometimes be satisfactorily sustained with the help of vasopressor drugs such as arterenol,[8,9] and necessary operative procedures carried out. Chronic perforation may present a bizarre picture. One of our patients was operated upon with a presumptive diagnosis of carcinoma of the head of the pancreas because of painless, progressive jaundice which proved actually to be due to compression of the common bile duct by a penetrating duodenal ulcer.

In the presence of obstruction operation is mandatory and should be carried out before severe depletion has occurred. Not infrequently obstruction of the pylorus will be found at operation to be of gastric rather than duodenal origin with the attendant possibility of malignancy.

Gastric ulcers may attain great size in the aged and be attended with marked debility. In one of our patients relief was long withheld because the patient was thought to be dying from inoperable cancer. Even at operation differentiation between benign gastric ulcer and carcinoma may be difficult. In one instance total gastrectomy was carried out because of the size of the lesion and the suspicion of malignancy. The possibility must always be kept in mind that any gastric ulcer may be malignant, in which event the patient's only hope lies in operation. For such reasons as these most gastric ulcers in the aged should be treated surgically. Complications of gastric ulcer should be dealt with in the same manner as complications of duodenal ulcer.

Jejunal ulcers, which usually manifest their presence in the aged by bleeding, should be treated by resection.

When surgical treatment is employed for peptic ulcer in the aged it should be adequate. Local excision of ulcers, ligation of bleeding points, pyloroplasties, and such, performed in an effort to spare the patient from a bigger operation often were followed by disaster or the need for subsequent operation which a more extensive procedure would have obviated. A patient who is bleeding should have a gastric resection, if he is operated upon at all. The same is true for the patient with intractable pain. Simple closure of perforation is probably safest, but it must be remembered that several of our elderly people subsequently required further operative procedures. Gastroenterostomy may suffice in the presence of obstruction, but even in the aged jejunal ulceration may follow. The fact that any operation at this stage of life entails added risk is all the more reason not to perform an inadequate one which may require subjecting the patient to the same risk again.

SUMMARY

A review of 135 patients over seventy years of age suffering from peptic ulceration and an analysis of the manner in which these cases were handled has been presented. Our feelings regarding the management of peptic ulcer in the aged have been stated. Medical treatment suffices for the majority of patients. The same indications for surgery exist as in younger patients.

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DISCUSSION

William B. Hutchinson (Seattle, Wash.): Dr. Schlicke has presented a timely and difficult subject very well. The fact that the patients in this series were often treated by physicians who were not specialists should be kept in mind. In a small series such as this each case is so important statistically. Surgery of peptic ulcer in the aged should not differ from that of any other age. They will tolerate surgery or its complications less well at age seventy than they would at age forty. It is quite comparable to the distinct difference in exercise tolerance of these two age groups. However, the seventy year old may on rare occasions outdo the forty year old physiologically and, therefore, each case should be evaluated individually.

There were seven deaths from acute hemorrhage, five medical and two surgical. Approximately three-fourths of these patients can be controlled immediately by non-operative management, provided it is pursued with a goodly quantity of blood. Many patients who die following non-operative management have received only a fraction of the blood which would have been required to replace the amount lost. After using approximately 3,000 cc. of blood administered reasonably rapidly and if there has been no response, we believe the non-operative treatment has failed and surgery should not be delayed. In those patients who are controlled and begin to bleed again surgery seems wisest.

Dr. Schlicke found that wound dehiscence was the precursor of death in four instances. This occurred in one-half the surgical deaths and he suggests greater precaution than the ordinary methods of closure, but did not enlarge on this point. We have for years adopted a method of closure in elderly poor risk patients whom we suspect are candidates for wound separation. I hesitate to mention this before such an august body, but it has been effective. This is the use of six or eight through and through silver wire sutures. No doubt many use this same method. None of us would hesitate to use this closure following wound dehiscence. We believe it gets the patient off the operating table rapidly and may prevent a future evisceration which may well be a fatal complication.

I agree with Dr. Schlicke in his condemnation of the local ligation of bleeding points, local excision of ulcers and pyloroplasties. For the elderly patient with complete or nearly complete obstruction and a low acid, gastroenterostomy is undoubtedly a good procedure with many happy results in the past. My own personal experience has not been entirely successful, however, and on two occasions following gastroenterostomy marginal ulcers have developed, one of which perforated. Subtotal resection is used, therefore, whenever possible, for many of these old folks will live long enough to develop the familiar complications of lesser procedures.

It has been a privilege to discuss this paper.

Frederic P. Shidler (Menlo Park, Calif.): I should like to discuss a portion of both Dr. Roberts' and Dr. Schlicke's papers as I believe they are interrelated.

It seems to me that as we get into peptic ulcer disease in the aged, there are two important factors that we should consider before any surgical operation is undertaken, namely, dehydration and renal function. We must remember that an aging person, especially one with an obstructing ulcer, is in chronic pain and probably vomits a good deal. His normal fluid intake is decreased because of his discomfort, so that unwittingly such a patient is found in marked dehydration and with a sodium depletion before surgical treatment is undertaken. Postoperatively, when the patient is in circulatory shock, or later has stomal malfunction, the blood potassium and sodium levels may be confusing because it is the first time that they have been tested. Dr. Moore of Boston especially has pointed out that it is the normal response to injury for serum sodium levels to drop and serum potassium levels to increase without any particular fluid loss. These effects are greatly increased if dehydration exists, so that a great many of our complications occur because we operate upon dehydrated patients before we know they are dehydrated. Secondly, renal function is so important because we know that electrolyte problems rarely exist if the patient has a pair of good functioning kidneys. We can avoid these pitfalls preoperatively if we give the patient some type of water tolerance test and demonstrate to our satisfaction that he is capable of excreting 750 to 1,000 cc. of urine a day.

Clarence Stafford (Los Angeles, Calif.): Dr. Schlicke has presented an excellent paper and emphasized to us the importance of recognizing and properly treating peptic ulcer in the aged. In a recent study made at the Los Angeles General Hospital it was found that approximately 40 per cent of the peptic ulcer cases were in patients over sixty years of age. In this older group one of four patients required surgery for some complication of ulcers with a resulting mortality of 20 per cent. This would further emphasize that peptic ulcer is not limited to young and middle-aged persons but that it presents one of the serious problems to be considered in the care of the elderly patient.

Rectal Infections and Ulcerations Associated with Blood Dyscrasias

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THE unusual and often severe infections and ulcerations of the rectum and anus which occur as complications of blood dyscrasias have been rarely, and then only cursorily, noted in the literature. Frequent reports have appeared, however, of such lesions involving the gums, soft palate, tonsils, pharynx, buccal and other mucosal surfaces such as the vagina, cervix uteri and intestinal tract.[2]

During the past four years, five bizarre rectal infections with hematopoietic abnormalities were seen in the private proctologic practice of one of the authors. In an effort to determine the incidence of this condition we undertook a review of cases of blood disease involving the myeloid elements which had been observed at the University of California Hospital. If 409 cases of leukemia, agranulocytosis, hypersplenism and aplastic anemia seen during a ten-year period, nine cases were involved with a severe rectal pathologic condition. This was an incidence of 2.2 per cent. Of 100 cases of anorectal abscesses and anal ulcers seen during the same period, 9 per cent were associated with blood dyscrasias. This latter high figure is of little significance, however, since operations for acute infections of the rectum at a hospital of the University type are infrequent. Likewise, the general over-all incidence of such lesions must be considered low. However, when unusual rectal infections are encountered, the possibility of myeloid aberrations must not be overlooked.

The clinical and pathologic findings in our fourteen cases are summarized in Table I. The differentiating characteristics as compared with rectal abscesses and ulcerations not associated with abnormalities of the blood, as well as the relation of these lesions to the primary pathologic condition, will be considered in more detail.

Some of the striking features in our cases, when contrasted to the more common rectal ulcerations and abscesses, were as follows: (1) the intensity of the pain; (2) the severity of the lesions; (3) the absence of formation of frank pus; (4) the rapidity of onset and local spread; (5) the "dirty" appearance of the ulcers which often became quite large; (6) the sluggishness or lack of healing; (7) the resistance to therapy, notably the poor response to antibiotics; (8) the recurrences; and (9) the gravity of the prognosis.

In this series of fourteen cases there were eight abscesses and five ulcers, while in one case a combination of both was present. Leukemia was the primary disease in nine cases, four resulting in abscess and four in ulceration; in one a lateral anal ulcer had given rise to an abscess. Of the leukemias, four were lymphocytic, three myelogenous, one monocytic and one undifferentiated. There were two cases of hypersplenism, both associated with ischiorectal abscesses. There was one case each of agranulocytosis and aplastic anemia with abscesses, and one instance of chronic hypoplastic neutropenia in which the entire perineum was involved in an ulcerative process. In thirteen of the fourteen cases no infections were present other than the rectal lesions.

Four patients (Cases I, III, X and XIV) had anal ulceration in leukemia, and there was one ulcer in a case of hypoplastic neutropenia with agammaglobinemia. In the latter case, which was the only one in this series in which infections other than the rectal ones occurred, the child had a persistent peri-auricular abscess and cellulitis with extension into the mastoid bone, together with gingival ulceration. All of the anal ulcers were extensive. They ranged in size from one-third the circumference of the anal canal (Case I) to diffuse cellulitis of the entire perineum with superficial necrotic ulceration.

TABLE I

Case No.	Age (yr.) and Sex	Diagnosis (Blood)	Diagnosis (Rectal Lesion)	Remarks
I	54,M	Acute monocytic leukemia	Anal ulcer involving ⅓ circumference posterior anal canal; spasm, edema, severe pain	Rectal lesion persisted until death
II	34,M	Acute granulocytic leukemia	Ischiorectal and supralevator abscess; sudden onset of pain; ragged 7 mm. orifice anterior anal canal communicated with abscess; cavity filled with necrotic debris; no frank pus	Incision and drainage; postoperative hyperpyremia, rectal hemorrhage; progressive induration and erythema; extended to right posterior ischium; wound margins became gangrenous; slough about anus; patient died
III	39,M	Myelogenous leukemia (plasmocytic) involving spleen	Anal ulcer extending into anal canal and over perineum to scrotum; indolent, dirty base with soft, waxy borders; biopsy: "perivascular infiltration compatible with leukemic ulcer"	Ulcer healed after splenectomy; long fistula from anorectum to scrotum persisted; fistulectomy with good result; patient survived
IV	51,M	Aplastic anemia	Sudden onset of rectal pain, chills, temperature 105°F.; spontaneous discharge from rectum of purulosanguineous material; in right anal canal was an orifice, 2 cm. in diameter, with necrotic borders, extending into perirectal space, discharging foul, brownish pink pus; induration of rectal wall extending 6 cm. proximally	Tract unroofed surgically; postoperative hemorrhage; wound persisted as pyogenic granulomatous ulcer until patient died
V	62,M	Subacute lymphatic leukemia	Onset with shaking chill, rectal pain, temperature 102°F.; right posterior perianal abscess containing 25 cc. thin, watery pus mixed with foul necrotic debris; roughened area 2 mm. in diameter, anorectal juncture; minimal induration about margins but not advancing	Incision and drainage of abscess; patient died
VI	61,M	Hypersplenism, pancytopenia	Ischiorectal abscess; 8 by 16 cm. cellulitis, right buttock; tissue of ischiorectal space black, necrotic, had not liquefied; few cc. of frank pus	Incision and drainage of abscess; splenectomy followed by rapid increase in purulent drainage of rectal wound, granulation and complete healing; patient recovered
VII	55,F	Hypersplenism, pancytopenia	Right perirectal cellulitis with fluctuant mass bulging into rectum; primary opening not found; drainage of considerable amount of black, liquid, necrotic fat and foul yellow pus; healing slow; abscess recurred; drained	Prompt healing of wound of recurrent abscess following splenectomy; patient recovered
VIII	46,F	Agranulocytosis (etiology?)	Left ischiorectal abscess 12 by 14 cm.; spontaneous rupture with drainage of watery, whitish necrotic material	Resection of colon for ulcerative colitis several years before; slow healing of abscess; patient recovered

TABLE 1 (*Continued*)

Case No.	Age (yr.) and Sex	Diagnosis (Blood)	Diagnosis (Rectal Lesion)	Remarks
IX	35,M	Acute lymphogenous leukemia	Right ischiorectal abscess containing 150 cc. greenish pus	Incision and drainage; slow but complete healing, which occurred during remission of leukemia while receiving aminopterin; patient survived
X	34,F	Undifferentiated leukemia	Anterior anal ulcer 1 cm. diameter with smooth border, necrotic base; posterior anal ulcer, 2 cm. in diameter, with bleeding base, overhanging, rolled edges	No response to therapy; marked progression of lesion to death 1 mo. after onset
XI	54,M	Chronic lymphatic leukemia	One month history of perianal pain; 2 days before entry spontaneous rupture of left lateral perianal abscess with discharge of yellow-green pus; recurred within few days; 50 cc. pus obtained	Incision and drainage; persistence of drainage with minimal evidence of healing at time of death 2 mo. later
XII	55,F	Chronic myelogenous leukemia	Left ischiorectal abscess; contained 60 cc. pus	Incision and drainage; wound clean with moderate drainage but unhealed at time of death 1½ mo. later
XIII	4½,F	Chronic hypoplastic neutropenia with agammaglobinemia	Diffuse cellulitis of anal canal and perineum with extensive ulceration	Persistent otitis, mastoiditis, facial cellulitis and gingivitis; patient died several days after appearance of anal lesion
XIV	88,M	Chronic lymphatic leukemia	One month history of foul, odoriferous, white mucoid discharge from right anterolateral fistula-in-ano; the fibrous sinus tract curved deep to sphincters and originated from a 1 cm. necrotic whitish gray anal ulcer 5 mm. above right anterolateral aspect of dentate line	Spontaneous drainage; persisted to present (4 mo.) without evidence of healing

In one instance (Case III) the diagnosis was plasmocytic myelogenous leukemia involving the spleen. In this case the ulcer, which was in the anterior midline, was a "dirty," indolent lesion with a grayish base and soft, raised, waxy borders. It extended up into the anal canal and anteriorly over the perineum to the base of the scrotum. Biopsy showed "heavy perivascular infiltration consistent with leukemia." Following splenectomy there was rapid, spontaneous healing. Three months later a fistula was found arising from a small anal ulcer at the site of the original ulcer. The fistulous tract coursed subcutaneously beneath the scar of the old perineal ulcer and emptied into a small fibrotic abscess cavity which had ruptured spontaneously. The ulcer and fistula were excised and healing was uneventful. At this time the blood abnormality was still in a state of remission.

In all of these cases the anal ulceration presented one of the most difficult problems in the care of the patient. Unlike the usual anal fissure or ulcer, pain was almost constant although it was severely aggravated by bowel movements. Anesthetic ointments were of little benefit, and warm compresses or sitz baths gave but partial, temporary relief. All of these patients were generally severely ill and the rectal distress compounded the difficulties of nursing care. For those who were bedridden, constipation presented an additional complica-

tion. Surgical excision was not attempted in any of these cases because it was believed that the underlying pathologic condition could not be removed, as in the case of fissure, and that the lack of wound healing would only result in extension of the process. Injectable, long-lasting local anesthetics would seem to have been useful, bearing in mind the calculated risk of additional infection. In general, these agents are to be condemned.

The nine abscesses were diagnosed as ischiorectal (Cases II, III, VII, VIII, IX and XII), supralevator (Case II), perirectal (Case V) and perianal (Case XI). In two cases the patient was suffering from hypersplenism, and following splenectomy the abscess cavities healed in a normal way. In the remainder, in which leukemia was present, there was progress or persistence of the abscess. Bacteriologic studies in five cases disclosed the causative organism to be Escherichia coli, Aerobacter aerogenes and non-hemolytic staphylococcus albus. The onset of an abscess sometimes occurred suddenly and was often evidenced by chills, hyperpyrexia and rectal pain.

When these abscesses were incised surgically or drained spontaneously, the material in most of them was not the thick purulent accumulation commonly found in abscesses in this region. The content was variously described as follows: watery, white with liquefied fat; foul-smelling, brownish pink; necrotic, non-liquefied; black, liquid, necrotic fat; necrotic debris; thin and watery; and frank, greenish yellow pus. The amount varied from a few cubic centimeters to 150 cc. when frank pus was present. In three instances (Cases II, IV and XIV) there were large intra-anal openings into the abscess as if an ulcer had eroded into the pararectal spaces. In one instance (Case XIV) the opening persisted and entered a deep, fibrotic sinus tract which curved about beneath the sphincters. Most of the abscesses involved extensive areas with cellulitis and extended peripherally for a considerable distance. In Case II the entire ischioanal fossa and the supralevator space lying above it had become a huge necrotic cavity. Following drainage of these abscesses the surrounding cellulitis did not resolve, or healed slowly. In some instances the skin at the borders of the site of drainage became necrotic, and secondary cutaneous infections occurred. Of interest, too, was the lack of general and local response to antibiotics.

In general, the severity of an infection depends upon (1) the type, number and virulence of the invading organism, and (2) the site and resistance of the host. These factors are normally in a state of dynamic equilibrium. The defense of the host is decreased in diseases such as leukemia, agranulocytosis, pancytopenia and aplastic anemia because of partial or complete loss of myeloid elements. Infection is most apt to occur in those regions where bacteria are usually present. When the first line of defense (namely the skin or mucous membrane) has been penetrated, infection becomes manifest. Granulocytes, produced by the myeloid tissue, are the most important elements in the mechanism of controlling infection. Jaffé[1] found that in the absence of myeloid matrix the bodily response of leukemic patients to infection is similar to that of patients with either aplastic anemia or agranulocytosis, in that the normal defense is lacking. There is less response to infection and the incidence of infection is higher in cases of lymphatic leukemia than in myelogenous leukemia.[3] Extensive ulcerations are known to occur as a result of leukemic infiltration as well as bacterial invasion.

The common anal fissure, or ulcer as it is more properly called, is usually a result of infection of an anal crypt followed by a series of events leading to a chronic, painful, localized lesion occurring predominantly in the midline, usually posteriorly, and exhibiting typical associated inflammatory reactions such as hypertrophic papilla, the "sentinel pile" and anal stenosis. It is amenable to conservative treatment or surgical cure. The atypical ulceration, on the other hand, shows no anatomic predilection and no associated "fissure triad." Operative therapy would seem to be inadvisable.

The common pararectal abscess, too, characteristically originates in an anal crypt with extension along well defined pathways into one of several cavities in the region. Treatment by adequate incision and drainage with, in many instances, ablation of the associated fistula, is definitive. The atypical abscess described here is quite likely due to direct invasion of the wall of the anorectum, in some instances by erosion of an ulcer. Limited anatomic spaces are, of course, involved; however, there is often extension beyond them by the cellulitis that has been unstemmed in its peripheral advance. While drainage or decompression was often performed in the leukemic abscesses, the infection did not

usually resolve. Postoperative hemorrhage occurred twice.

SUMMARY AND CONCLUSIONS

1. Fourteen cases of anorectal ulcerations and abscesses occurring in patients with blood dyscrasias are reported.

2. The lesions presented bizarre clinical manifestations in comparison to their counterparts in patients without abnormalities of the blood.

3. Although anorectal abnormalities of this type are uncommon, the diagnosis of a myeloid aberration is to be considered when such lesions are encountered.

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DISCUSSION

Edwin G. Clausen (Oakland, Calif.): Although we have been aware of ulcerative lesions associated with blood dyscrasia, the rectal lesions have not received much attention. Drs. Birnbaum and Ahlquist have shown us the severity and importance of such lesions. When reviewing their cases one is impressed by the fact that none of the lesions healed unless the dyscrasia was corrected or underwent remission. Any attempts at definitive surgery during the height of the disease obviously would be harmful. In those instances when splenectomy is curative or will precipitate remission, the problem is less severe, although the general condition of the patient may dictate the time of splenectomy. The use of cortisone and ACTH may be helpful in precipitating remission. Experience has shown that the presence of infection is no contraindication to the use of cortisone or ACTH in the management of acute agranulocytosis where the response is rapid and the use of the drugs is of short duration. The use of cortisone and ACTH in the leukemias is less dramatic and of value chiefly in the acute forms and especially the lymphatic type. When used in these blood dyscrasias associated with infection one might expect remission of the disease and suppression of the inflammatory process. However, the infection may proceed perhaps with increased vigor unless the organisms are susceptible to specific antibiotics. The authors have indicated that antibiotics are not very effective. In the management of the leukemias cortisone must be continued for a prolonged period; hence the local inflammatory lesion could conceivably get out of hand, particularly in those cases in which remission is not achieved. However, in the authors' series death ensued in all cases in which remission did not occur. I have had experience with one patient with aleukemic leukemia in whom an ischiorectal abscess developed. He was treated with cortisone and ACTH; and although there was no remission from the disease, through the use of large doses of streptomycin and penicillin the abscess cavity gradually cleared and a small fistula was the final result. I would like to ask Dr. Birnbaum if he has had any experience with the use of cortisone and ACTH in the management of such cases.

Russell Klein (San Rafael, Calif.): Both authors are to be congratulated on their fine paper dealing with this somewhat unusual condition. I have not had the opportunity of seeing as many cases as they have reported, but I am certain my results have been no better.

It is most distressing to have so little to offer these patients from the surgical standpoint. Simple incision and drainage is all that I have ever attempted and this has had little apparent influence on the course of the disease, as all patients have died in a short period of time.

There are two points which I would like to bring up for discussion. First, with rapid advances in chemotherapy it is now possible to produce remission where previously this was most unusual. Dr. Byron Hall of San Francisco, who has had a great deal of experience with blood dyscrasias, informs me that it is now possible to induce remission in over 60 per cent of cases of acute leukemia, whereas previously the expectation was about 10 per cent. Such a remission may occur in a matter of several weeks and may last for as long as fifteen months. Certainly all patients should have supportive treatment, including the adequate drainage of infection while chemotherapy is being tried. If remission is produced, resistance to infection approaches normal and is usually adequate to overcome the local inflammatory process.

My second point is the theoretic desirability of short-circuiting the fecal stream during the acute inflammatory process. While these patients are usually quite ill, have the authors considered this possibility in their series of cases?

Walter Birnbaum (closing): All of the patients in our group were under the care of hematologists or internists. No attempt has been made in our paper to describe in any detail the measures that were used in combating their leukemias, but treatment included most of the chemotherapeutic substances as well as cortisone and ACTH. The results were variable and, as Dr. Clausen has

mentioned, the appearance of the rectal lesion paralleled the improvement or deterioration in the blood picture. The lack of response to the antibiotics was a striking feature in this series and the difference between the *in vivo* and *in vitro* reactions of the organisms is a provocative phenomenon.

No attempt was made to perform a diverting colostomy because in each instance it was believed that surgery should be held to a minimum. The complications of poor healing, infection and hemorrhage were thought to be contraindications for any but the most minor and urgent operations.

The therapy of these blood dyscrasias was, of course, not dwelt upon since it was the purpose of this report to present a condition which previously had had no formal recognition, to emphasize the gravity of such a complication, and perhaps most important, to bring to your attention the advisability of having blood studies made when such unusual rectal infections are encountered.

The Traction Sigmoidoscope

DEVELOPMENT AND PRELIMINARY REPORT

A. E. Moore, M.D., *San Diego, California*

The transverse and sigmoid portions of the colon both have a mesentery and are flexible. The traction sigmoidoscope has been developed to utilize the inherent flexibility of the sigmoid gathering it down over the sheath with the result of sliding the bowel onto the scope.

As early as the fifth century an instrument similar to the later named proctoscope was used.[1] In 1852 Desormeaux of Paris introduced

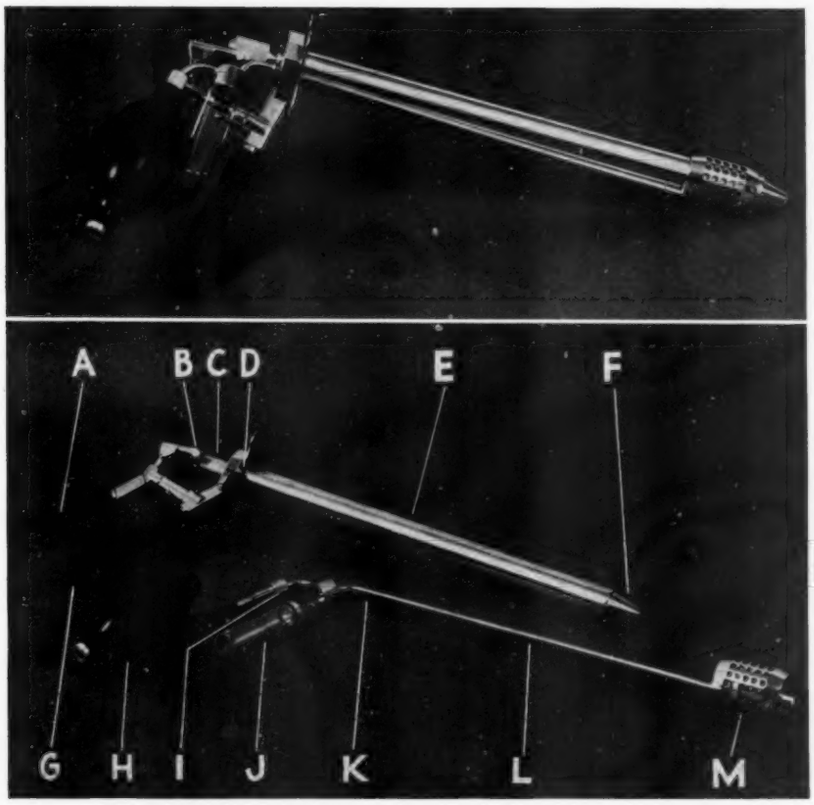

Fig. 1. Traction scope assembled.

Fig. 2. (A) light cord; (B) light; (C) obturator handle; (D) slot; (E) sheath; (F) obturator; (G) handle; (H) rubber suction bulb; (I) release valve; (J) mucus trap; (K) stop collar; (L) suction tube; (M) suction sleeve.

of the scope, similar to the gathering of a curtain over a curtain rod.

Figures 1 and 2 show the instrument. Figure 3 illustrates the limit to which a 25 cm. scope can be passed in a normal sigmoid colon. Figure 4 is a drawing which shows the effect of pulling the activated traction sleeve downward and named the endoscope.[1] In the year 1895 Kelley of Baltimore described the first practical sigmoidoscope.[2] During the past two generations signoidoscopic examination has been improved by the indirect methods of air insufflation and knee-chest or similar position. The traction scope now adds a direct improvement

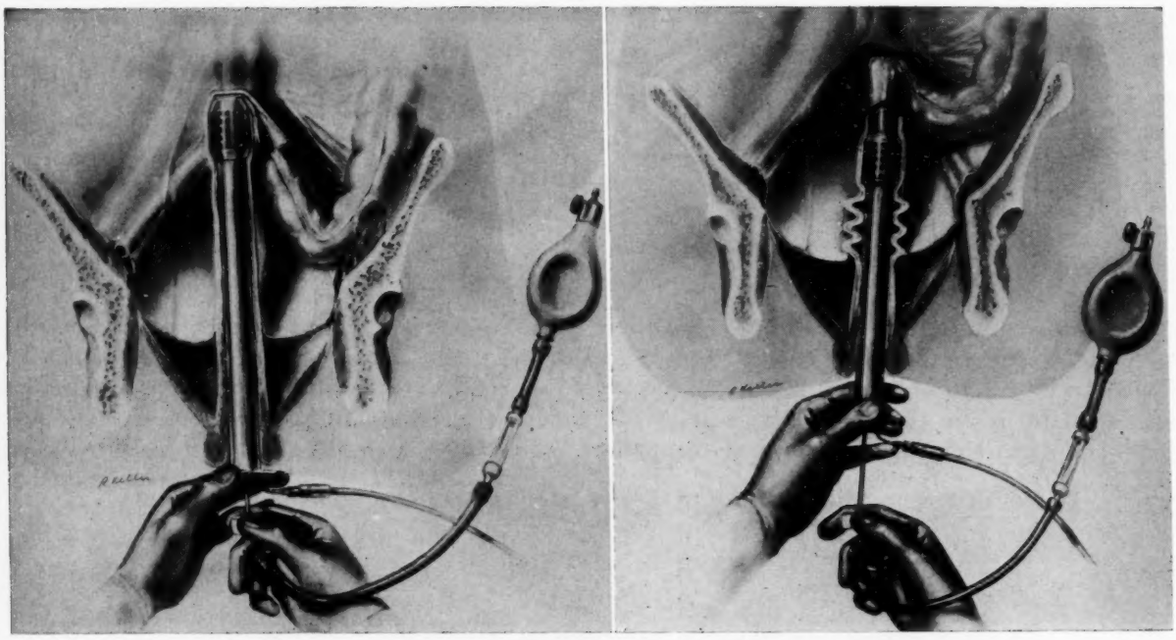

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FIG. 3. The end of a sigmoidoscope passed for 25 cm. will be in front of the lumbar vertebra and out of the pelvis. In the normal average, this point is the distal flexure of the sigmoid colon. A push on the scope simply stretches that part of the bowel between the anus and the distal flexure. Not only is there no gain, but there is also danger of perforation. The proximal bowel is acutely angulated down toward the proximal flexure.

FIG. 4. Withdrawing the scope 4 to 6 cm. and redirecting it toward the left side relieves the tension on the bowel and permits a normal traction pull.

to the instrument and increases the length of bowel that can be reached for diagnosis and treatment.

After two unsuccessful preliminary models had been made, the third scope was completed in April, 1954. Immediately thereafter a freshly removed operative specimen of the rectum and low sigmoid colon was available for testing. When the scope was passed into this specimen and 15 inches of vacuum applied to the suction ports, a good grasp of the mucous membrane was obtained. It was found that under these conditions the bowel could be moved backward and forward at will over the scope. The first patient in this series had been previously scheduled for removal of a polyp in the sigmoid colon. On May 3rd the patient was placed in the lithotomy position after anesthesia and the scope was passed as far as possible; suction contact was then made and by pulling down the bowel the polyp came into view. It was removed through the scope with a snare. While the frozen section was being prepared an x-ray film was made. (Fig. 5.) Preoperative film of the polyp is shown in Figure 6. Figures 7 through 11 show five subsequent patients upon whom polypectomy was performed through this instrument.

The first successful traction scope was made, by accident, with a sheath of 1 inch outside diameter and the traction sleeve 1¼ inches. While this is perfectly satisfactory and even desirable for anesthetized patients, there are few adults who can normally tolerate the passage of a 1¼ inch instrument. Late in May, 1954, another model was received; this measured ¾ inch diameter sheath and 1 inch on the sleeve. During the previous three-week period there had been numerous conjectures as to whether or not the application of suction to grasp the mucous membrane of the bowel would or would not be painful. This new scope was passed to 14 cm. on a man who had had an anterior resection. Five inches of vacuum was applied using power water suction and the patient denied any sensation of pain or discomfort. It was then discovered that the suction system had abruptly risen to 15 inches of vacuum. Suction was released and upon inspection the mucous membrane looked as though it had developed measles. Every one of the ⅛ inch diameter contacts was com-

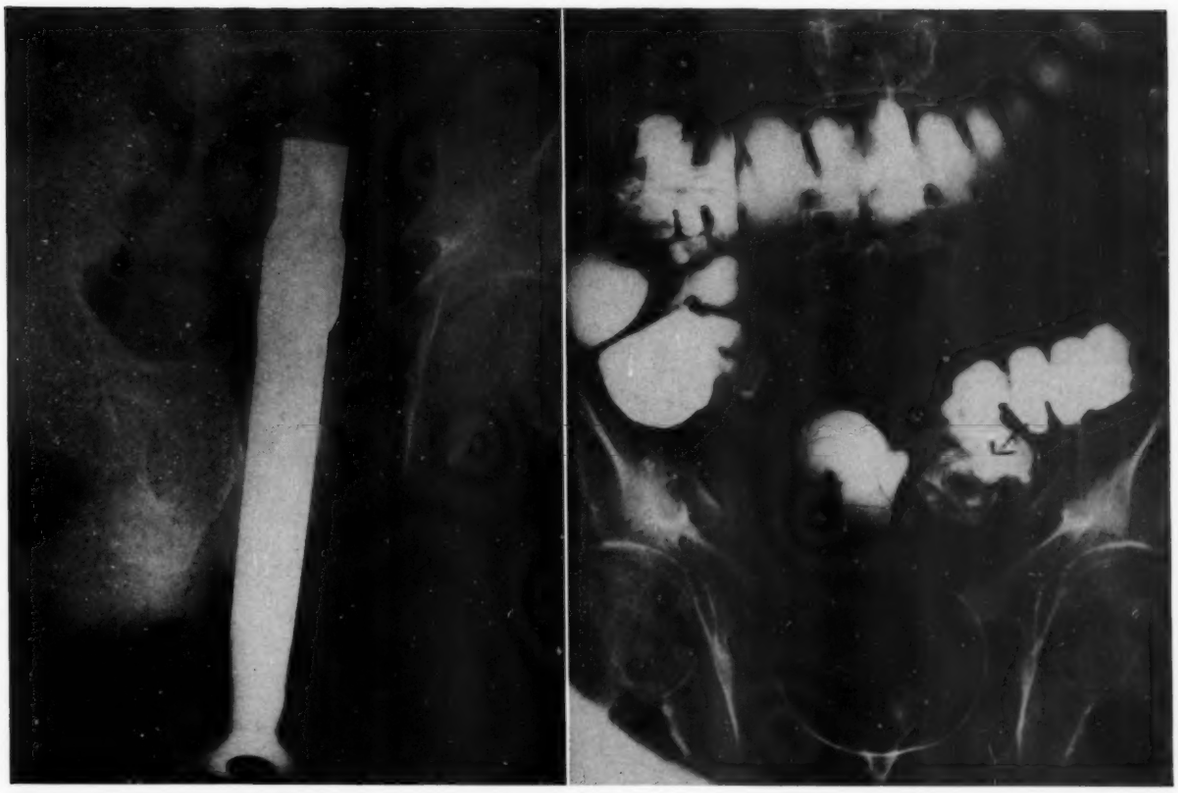

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FIG. 5. The traction scope is shown in the first patient upon whom it was used; inserted for 20 cm., and the polyp was 27 cm. as shown by measurements when the abdomen was open. See preoperative x-ray. (Fig. 6.)

FIG. 6. Patient C. R., sixty-nine year old man (May 3, 1954). Polyp removed in the hospital under anesthesia with a snare. Frozen section conclusively demonstrated carcinoma. Immediate resection was done. At operation a rectal catheter was passed up to the palpable base of the pedicle and measurement was 27 cm. above the anus.

pletely hemorrhagic. Ten days later this patient was again scoped, no residual whatsoever noted, and he denied any passage of blood.

At this writing ten different scopes and fifteen different traction sleeves have been made. Each is tested on freshly removed pig's intestine (Cudahy) using 5 inches of mercury vacuum and a spring scale. The scope assembly is pulled and the traction required to counteract the suction grasp is measured in ounces. (Fig. 12.) Scopes that pass the preliminary tests are then used on patients under anesthesia following various surgical procedures such as thyroidectomy, breast biopsy, cholecystectomy and hernioplasty. All of the early experimental work on patients was done using water suction and a vacuum meter. It was soon found that 10 inches of vacuum invariably produced mucosal hemorrhage while 5 inches of vacuum often did not cause bleeding. It was also determined that less than 20 ounces of pull was not effective in grasping the bowel. This led to the various modifications of the traction sleeve each of which had to meet the requirement of using only 5 inches of vacuum yet be capable of pulling 20 ounces or better. The traction scope now has safety factors built into the design so that the suction grasp and pull is not strong enough to make a perforation on fixed bowel and mucosal hemorrhage is always minimal because no more than 4 inches of vacuum can be produced by the suction bulb.

Figure 13 which we have been calling the lateral knee-chest position has more or less evolved during the development of the traction sigmoidoscope. It is a position into which the patient can readily and easily be placed on any level table while under anesthesia. It provides a large table space for instruments and permits the operator to stand comfortably. The lithotomy position was found to be very poor because the operator had to practically sit on the floor and exposure of the bowel was not good. Sim's position was likewise unsatisfactory as the left

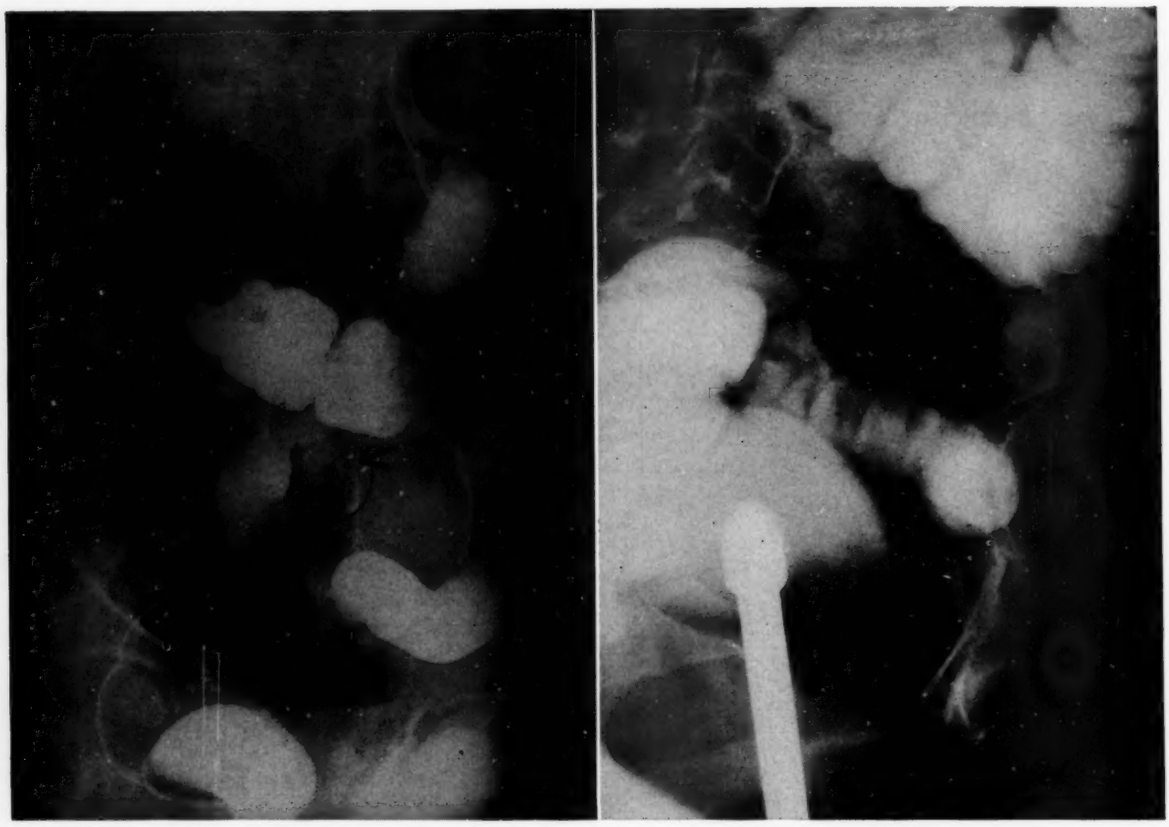

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FIG. 7. Patient A. H., forty-one year old man (June 14, 1954). A microscopically benign polyp on a long pedicle was removed in the office with a snare. The level was somewhere above 25 cm. Subsequent barium enema was normal and sigmoidoscopic examination above 25 cm. was normal on two occasions.

FIG. 8. Patient C. B., thirty-two year old woman (August 13, 1954). In the hospital under anesthesia, the base of the polyp was ligated and then divided with a snare. Permanent sections showed carcinoma. Resection was done four days later. The distance from the anus to the base of the pedicle was again measured by passing a rectal catheter from below when the abdomen was open and was 26 cm. No residual carcinoma.

thigh was in the way of an operator trying to pass an instrument under direct vision. The Bouie type position has the disadvantage of indirectly limiting the flexibility of the sigmoid by increasing the intra-abdominal pressure as the patient lies on the abdomen relaxed under anesthesia. This could probably be corrected by the use of a special table.

Without anesthesia and in knee-chest or similar position the introduction of a scope through the sphincter allows air to enter the rectum and colon. For the traction scope to make suction contact in this position, it is necessary to obstruct the open base of the scope with the thumb or with a lensed stopper and evacuate the air with two or three pumps of the suction bulb. Experience without anesthesia is too scant to draw conclusions, but as others are now testing this scope, its effectiveness will soon be a matter of independent record. It should be emphasized that all scopes including the traction scope should be passed only under direct vision and never blindly.

The traction scope has been used on over 100 patients. Only preliminary statistics are available and these include examinations made with scope models that have since been discarded. Occasionally two models were tested on the same patient. Seventy-four patients have been scoped under anesthesia. In 57, or 75 per cent, more than 25 cm. of bowel has been exposed. The remaining seventeen patients in whom less than 25 cm. of bowel has been reached are examples of the limitations of any sigmoidoscope as in all of these the scope could not be passed to the distal flexure.

Shatz and Freitas[3] have determined the extent of examination on the average patient

9 10

FIG. 9. Patient L. D., fifty-seven year old woman (September 10, 1954). In the hospital under anesthesia the scope was passed 4 cm. above the site of a previous colon resection for carcinoma. The polyp pedicle was ligated and removed with a snare. Pathologic report revealed adenoma malignum with no tumor in the pedicle. "Resection was not done as patient has liver metastases proved during her other operation."

FIG. 10. Patient V. H., fifty-eight year old woman (September 28, 1954). The polyp pedicle was ligated and resected with a snare. Pathologic report revealed "carcinoma without involvement of the pedicle." Resection was done four days later. A catheter passed up to the base of the polyp measured as 34 cm. No residual carcinoma was found in the resected specimen.

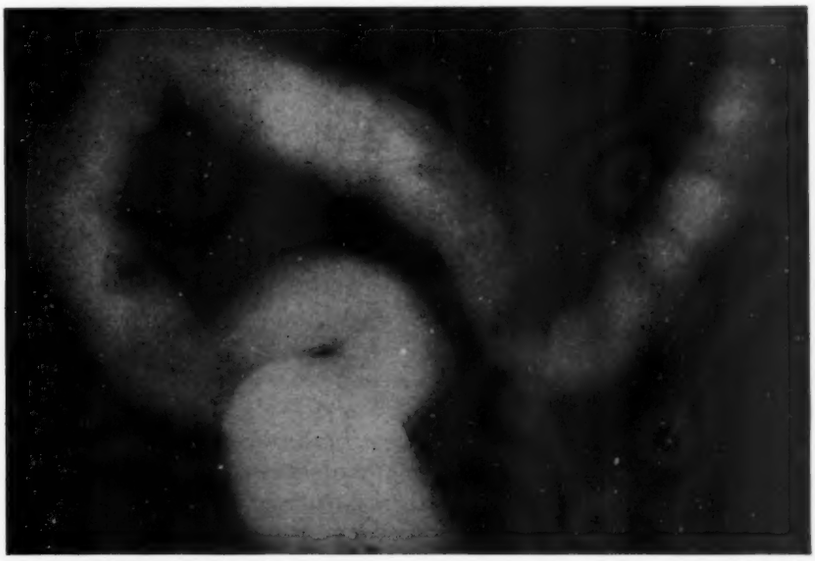

FIG. 11. Patient R. G., fifty-seven year old woman (September 29, 1954). The polyp was removed by ligation and a snare. A polyp of the cecum was then removed by laparotomy and colotomy. The level of the sigmoid polyp pedicle was 35 cm.

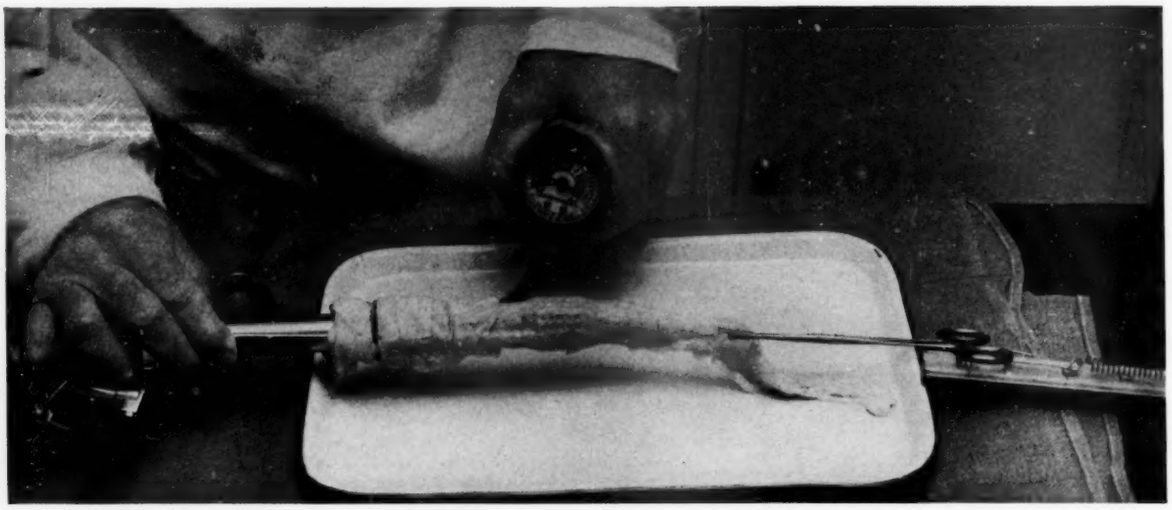

FIG. 12. The rectum is cut away from the freshly removed pig's bowel and after passing it over the scope a rubber band is applied to substitute for a sphincter and seal off air leakage. Five inches of mercury vacuum is applied by mechanical suction and the pull necessary to break the suction is measured on the spring scale which is graduated in ounces.

in knee-chest position using a 25 cm. scope. Scattergram A (Fig. 14) shows the normal average while scattergram B is a composite of the films shown in Figures 6 through 11.

In one patient, M. G., a fifty-one year old woman, a sigmoid colon polyp could not be

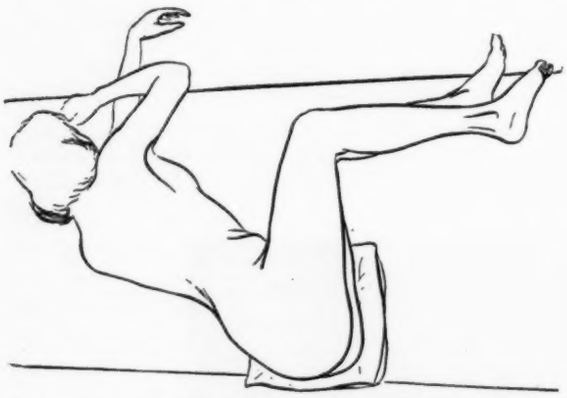

FIG. 13. Lateral knee-chest position. The patient lies on the left side with the back extended, hips and knees flexed. There is a 2 to 3 inch thick small pillow under the hip. Those of us who know the rail fence can better understand this position by considering the feet on the bottom rail, the thighs resting on the second rail and the third rail keeping the back arched.

reached. On sigmoidoscopic examination* the bowel was found to be fixed and the instrument would not pass beyond 18 cm. Laparotomy was performed and the pelvis found to be virtually frozen from previous operative adhesions. The sigmoid colon was freed after much tedious dissection and through colotomy a benign polyp was removed from the proximal flexure.

Aside from the rather common superficial mucosal hemorrhages and one instance of a 5 mm. laceration in the mucous membrane, there has been only one misadventure while developing this instrument. A traction sleeve was designed with transverse slots instead of circular holes and when tested on pig's bowel was noted to pull 40 ounces. When passed on a patient and a traction pull made, it was very effective; but when the suction was released, the bowel remained caught. An attempt was then made to withdraw the entire assembly but it could not be extracted. Air was blown into the suction tube but did not effect a release. After a trying few minutes it was found that when the scope was pulled down a finger passed alongside could be made to reach the sleeve for manual disengagement. The scope was immediately re-inserted without the sleeve to determine the extent of damage and it was quite surprising to note that the site of locking of the mucous membrane to the suction sleeve could not be detected. The patient has had no complaint whatsoever referable to this procedure. Needless to say this mishap cannot occur with the latest model traction sleeve.

Failures. As originally predicted the traction sigmoidoscope will not function as a pull instrument without flexibility of the sigmoid. Conditions which have been noted to produce fixation are: (1) postoperative adhesions, (2)

* Performed in October, 1954.

diverticulitis, (3) carcinomatous stricture, (4) previous pelvic inflammatory disease, (5) short sigmoid mesentery, (6) fat and heavy mesentery and (7) some previous colon resections. In addition the instrument has not functioned because of colitis with fibrous tissue deposit in the wall of the bowel creating a semi-rigid sigmoid colon. Other limitations are those which apply to any proctoscopic examination.

Contraindications. There are two contraindications, namely, proctitis or colitis with ulceration or inflammation of the mucosa; and purpuric states with a tendency to bleed (theoretic).

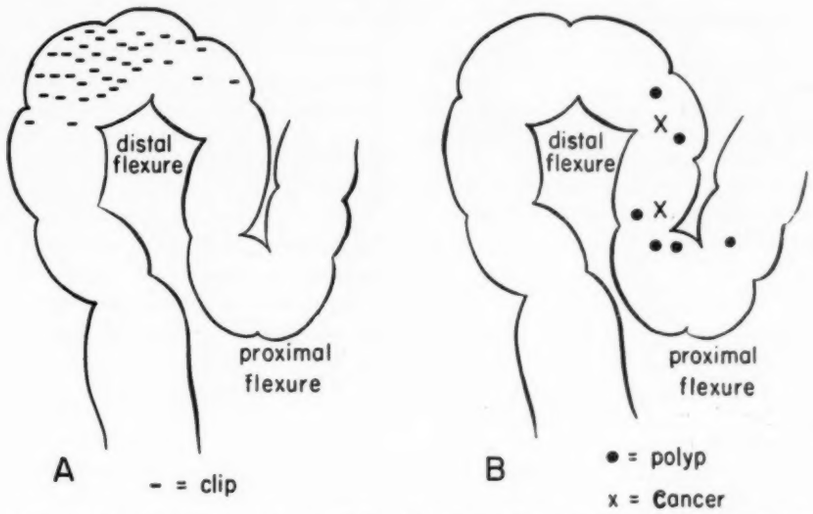

FIG. 14. Scattergram A is reproduced by permission of Shatz and Freitas.[3] A metal clip was applied to the mucosa during sigmoidoscopic examination in knee-chest position on thirty-seven patients and then localized by x-ray. This composite shows that average examination reached the distal sigmoid flexure. Scattergram B represents six patients with polyps and two with carcinomas. Each polyp was exposed and removed through the traction sigmoidoscope. Both carcinomas were exposed prior to resection and one was biopsied.

SUMMARY

1. Ten different working models of the traction sigmoidoscope have been made and t sted on freshly removed pig's bowel.

2. Promising models when used on patients have shown that (*a*) superficial mucosal hemorrhages are infrequent when 5 inches of mercury vacuum or less is used; (*b*) 5 or 15 inches of vacuum applied to the mucosa produces no sensation whatsoever in the fully conscious patient; (*c*) severe mucosal hemorrhage from 15 inches of vacuum completely healed in ten days; (*d*) 20 ounces of traction are necessary to grasp and properly manipulate the normally flexible colon; (*e*) 20 to 24 ounces of traction grasp will slip on rigid or fixed bowel. Perforation by traction cannot occur because of this built-in safety factor.

3. Polypectomy has been performed on six of seven patients when the polyp was completely beyond reach of the ordinary scope.

4. Under anesthesia about three of four patients can be scoped for 35 cm. or more.

5. The curtain rod principle of this scope becomes non-operative on a fixed sigmoid colon.

6. Benign polyps were discovered in eight of the seventy-four patients in the series, or about 10 per cent.

CONCLUSIONS

An improved sigmoidoscope has been developed which under proper conditions permits examination, biopsy and polypectomy in the heretofore unreachable proximal sigmoid colon flexure. This is the first combination push and pull endoscope. Its importance is now being determined by independent evaluation.

Sigmoidoscopy under anesthesia is simplified by the position of the patient which has evolved during the course of this work and which we call, for the lack of a better name, the left lateral knee-chest position.

Acknowledgment: The fabrication of experimental models, the engineering ability and technical assistance of the Surgical Instrument Division of The Technical Oil Tool Corporation have been invaluable in the development of the traction sigmoidoscope. Photography was done by Mr. Howard E. Jope.

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DISCUSSION

Robert A. Scarborough (San Francisco, Calif.): Dr. Moore deserves high commendation for his conception of the principle for a traction sigmoidoscope, and for his mechanical ingenuity in perfecting an instrument of well engineered efficiency. His experience with the instrument has demonstrated its usefulness.

Dr. Moore has been kind enough to supply me with one of his instruments for trial, and bold enough to invite criticism.

Trial has so far been limited to examination of eight patients in the past two weeks, as an office procedure without anesthesia. This preliminary trial has demonstrated that in some patients, at least, direct visual inspection of the sigmoid can be attained to a higher level than by any other previous instrument.

I can offer no criticism of an instrument of such obvious value. However, I would like to comment and caution about its use.

In diagnosis, the usefulness of the traction scope is in the discovery of neoplasms above reach of the conventional sigmoidoscope. The latter instrument is sometimes available but seldom used in far too many doctor's offices. It is obvious that this new instrument will not increase the utilization of sigmoidoscopic examination by the average physician. It will be of some value to the doctor who already utilizes the conventional scope efficiently.

Barium-air contrast enema examination by a competent roentgenologist gives reasonably satisfactory diagnosis of neoplasms in the sigmoid and the rest of the abdominal colon. Traction sigmoidoscopy is not a substitute for such examination.

X-ray diagnosis of a polyp of the sigmoid is less accurate under the identical conditions which will prevent successful use of the traction scope, namely, fixation of redundant loops of sigmoid by adhesions from any cause, and particularly fixation resulting from diverticulitis.

Dr. Moore has demonstrated his ability to remove polyps high in the sigmoid through his instrument. I believe that he considers this its principal value.

I am reluctant to share his present enthusiasm for two reasons: (1) The hazards of perforation, hemorrhage or rapid development of a hematoma which may be unrecognized or uncontrollable through the scope. (2) My conception of polypoid disease of the colon as a condition in which there is a high frequency of multiple primary tumors.

I believe that the presence of a single polyp of the abdominal colon, demonstrable by x-ray study, is a definite indication for abdominal exploration of the entire colon. Thus it is not uncommon to discover other unsuspected polyps, or even a frank carcinoma.

In our own earlier experience, multiple primary lesions occurred in one of five such patients. More recent experience indicates a frequency of multiple lesions in two of five cases.

I hope that my remarks will not be taken as criticism of Dr. Moore's traction sigmoidoscope. I am sure that further experience will more clearly define both its usefulness and its limitations.

Alois E. Moore (closing): In closing I would like to thank Doctor Scarborough for his most generous remarks. It is the use of this instrument by others that will eventually determine its place in the diagnosis and treatment of sigmoid colon disease.

The Implications of Testicular Cytology in the Treatment of Cryptorchidism

FRANK HINMAN, JR., M.D., *San Francisco, California*

From the Department of Surgery-Urology, University of California School of Medicine, San Francisco, California.

THIS study was undertaken in an attempt to determine the age at which the cryptorchid child should be treated.

Although 10 per cent of newborn children have undescended testes, four-fifths of these children are normal by the time they reach puberty. Only one adult man in 500 is cryptorchid. However, it is well known that if the testes remain within the abdomen until the age of twelve or more, they are irreversibly damaged by the higher temperature. What has been less well accepted is the fact that serious injury may be sustained before puberty. The purpose of this paper is (1) to demonstrate by a cytologic study of biopsies that retained testes are significantly retarded before puberty when compared with normal testes,* and (2) to present the implications for surgical correction. In addition, it will be pointed out that many retained testes, especially if unilateral, are themselves fundamentally abnormal, so that the surgeon should not blame his technic if the testes do not function.

Biopsy Material. Study was made of thirty testicular biopsy specimens taken during the course of routine orchiopexy at the University of California Hospital since 1948. With few exceptions the specimens were fixed in Bouin's solution to prevent cytologic distortion, and stained with hematoxylin-eosin. This material was then compared with testicular specimens from children of comparable ages obtained after death, avoiding cases in which there were prolonged illnesses.

Cytologic Differences of the Retained Testis. Before six years of age few differences can be noted between the normal and the retained testis. In the testis of a normal three year old child (Fig. 1A) the testis cords have grown in length but the diameter has not increased. Comparison with a biopsy of the retained testis of a three year old child (Fig. 1B) shows no significant difference.

Little testicular development occurs until the end of the sixth year. Then the tubules gradually enlarge in diameter by increase in syncytial cytoplasm, and more and more tubules acquire lumens. Increasing numbers of germ cells appear. In the normal testis of a child aged nine years (Fig. 2A) the tubules have enlarged and the germinal epithelium has developed two good layers. In contrast, a section from the intra-abdominal testis of a child of the same age (Fig. 2B) shows that the tubules are obviously smaller with much more loose stroma, and the germinal epithelium itself is more immature.

By twelve years adult forms are beginning to appear in the germinal epithelium and the number of spermatogonia has greatly increased. The testis of a normal twelve year old boy (Fig. 3A) may be contrasted with a retained testis from a boy of the same age with cryptorchidism. (Fig. 3B.) Note how the germinal layers in the retained testis are poorly developed, and few if any of the spermatogonia are maturing.

At 16 years the normal testis (Fig. 4A) has differentiated completely. However, the retained testis shows not only retardation of development but also actual destructive changes in the germinal epithelium. (Fig. 4B.) These alterations are irreversible.

Congenitally Abnormal Testis. Testes which are fundamentally abnormal form an important group, separate from those in which maturation has been delayed by high intra-abdominal temperature. Abnormality is especially frequent in unilateral cryptorchidism: the fact that the one testis has descended indicates that pituitary stimulation is adequate and that the other testis is abnormal either in formation or position in canal. The remnant contains only a

* For more detailed studies of the cytology of retained testes, the reader is referred to the work of Cooper[1] and of Robinson and Engle.[2]

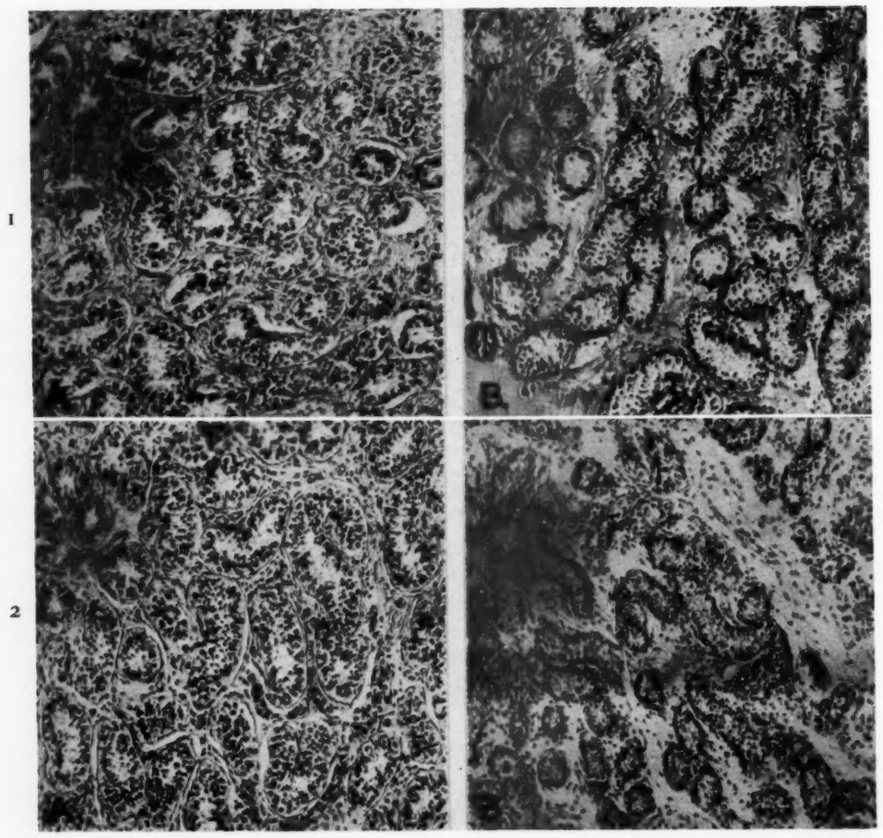

FIG. 1. A, normal testis at three years. Immature testis cords. B, cryptorchid testis at three years. Not significantly different from normal testis.

FIG. 2. A, normal testis at nine years. Tubular diameter is increased and lumens have appeared. The germinal cells are more differentiated. B, retained testis at nine years. Note the immaturity of the germinal epithelium and the persistence of tubules of small diameter. Increase in intertubular connective tissue.

few distorted conducting structures and clumps of Leydig's cells. A testis almost as hypoplastic is shown in Figure 5. It, however, had reached a normal scrotal position, so that increased temperature could play no part in its destruction. Although these are admittedly extreme examples, many lesser degrees of abnormality occur. Such abnormalities are the reason for many of the failures which are attributed to surgical treatment.

COMMENTS

Since Cooper's[1] pioneer work in 1929 it has been known that the typical undescended testis is fundamentally normal but becomes retarded and finally damaged by its retention. Many physicians have hesitated to treat these patients actively however, because of the appreciable proportion who are relieved spontaneously. Those testes which will not descend spontaneously must be distinguished from those which will descend, and the distinction must be made before the higher intra-abdominal temperature can produce serious retardation.

The actions of administered gonadotrophic hormone on the mechanisms for testicular descent are equivalent to those of the hormone released from the child's own pituitary gland at puberty. Since the side effects of the administered hormone are reversible and harmless, this substance should be given early before irreversible changes can occur in the retained testis. Operation need not be deferred if treatment with hormone fails, since lack of response to hormonal therapy indicates to the surgeon that the testis would not descend at puberty. The dangers and difficulties of operation are little different in children of six than in those twelve years of age.

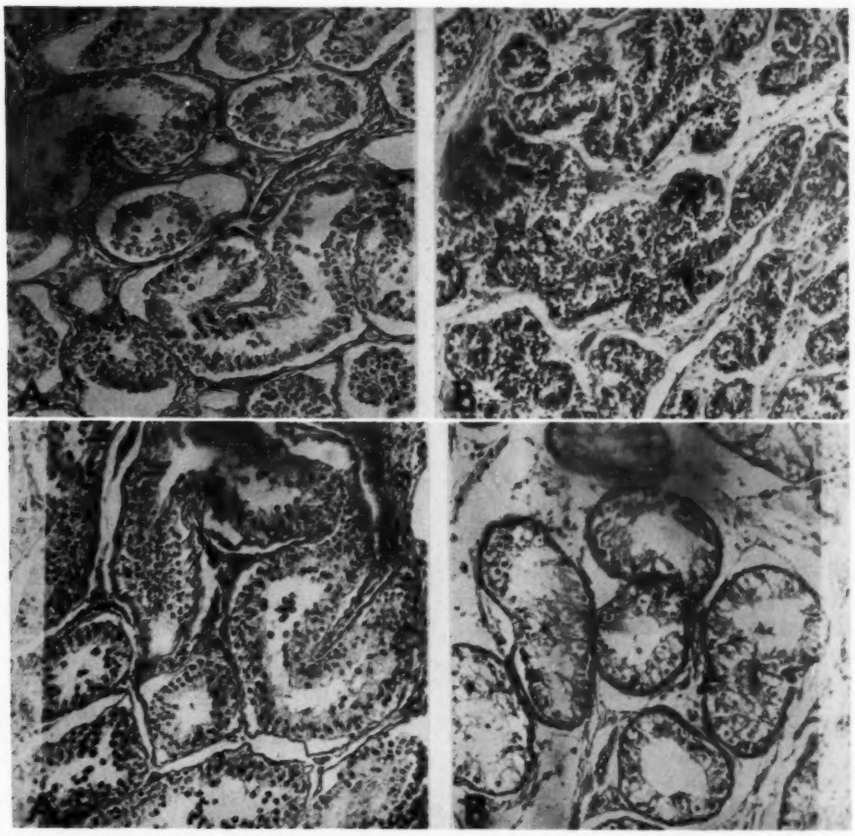

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FIG. 3. A, normal testis at twelve years. Adult tubular diameter has almost been reached and the germinal epithelium looks almost mature. B, cryptorchid testis at twelve years. Small tubules with immature, almost pyknotic epithelial cells. It is apparent that this is a seriously damaged testis.

FIG. 4. A, normal adult testis (seventeen years). Slight increase in tubular diameter and maturation (compare with Figure 3A). Spermatogenesis is now occurring. B, retained testis at seventeen years. Disorganized and damaged germinal epithelium with completely absent spermatogenesis.

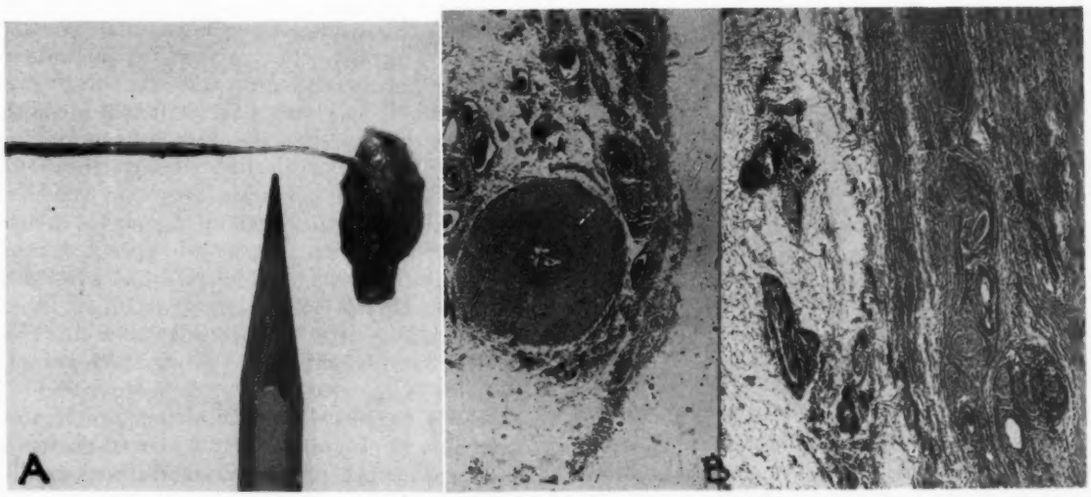

FIG. 5. A and B, gross and microscopic views of hypoplastic testis, epididymis and was removed from scrotum of patient eight years old in whom no gonad was palpable on that side.

Certain general rules, then, will help the surgeon confronted with a cryptorchid child to know what procedure must be done and at what age.

1. All hormonal and surgical procedures should be carried out before the child is six years old, to avoid thermal damage to the testis.

2. If a hernia is present, the problem is simple (as Snyder and Chaffin[3] have indicated). Before the child reaches the age of six, the hernia is repaired and the testis brought into the scrotum.

3. If bilateral cryporchidism exists, a trial of anterior pituitary-like hormone (1,000 units every other day for two weeks) will bring the testes down if they are capable of doing so at puberty. If descent does not occur, bilateral operation is indicated.

4. In unilateral cryptorchidism a trial period of hormone stimulation should be instituted. It will not be harmful and will make the operation technically easier if descent does not occur. The testis is abnormal in position or formation and probably will not respond. Orchiopexy, or if that is not possible, orchiectomy, should be performed if hormonal therapy is ineffective.

SUMMARY AND CONCLUSIONS

Testicular biopsies from thirty patients with cryptorchidism, when compared with sections of normal testes from children of similar ages, showed definite retardation in the specimens obtained at or after nine years. Maturation is retarded, however, even in the three preceding years. Treatment should be completed before these changes can occur.

The undescended testis associated with a clinically apparent hernia should be operated upon before the child is six years of age without further delay. The bilaterally undescended testes often will descend after administration of gonadotrophin; this should be tried before the child is six years of age. If this hormonal therapy fails, however, bilateral operation should be performed at once. The unilateral undescended testis, although probably not susceptible to hormonal stimulation, should be tested with gonadotrophin before the child is six years of age and then operated upon soon thereafter if descent does not occur.

This program will afford the greatest chance for fertility and should not increase morbidity and mortality.

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DISCUSSION

Leon Goldman (San Francisco, Calif.): In the past, decisions as to the specific age at which the undescended testis should be surgically brought down into the scrotum were loosely contrived; the precise prepubertal time for surgery was often determined by the physician's intuition or "clinical experience." Anterior pituitary gonadotrophin might be injected in varying dosages for periods of time in order to provide "trial stimulation" for non-surgical descent of the testis.

I wish to re-emphasize Dr. Hinman's observation that if hormonal therapy fails to cause testicular descent, the physiologic impetus of the patient's pituitary secretion at puberty will not be effective and operation should be carried out before irreversible changes occur. There is nothing more frustrating to the surgeon than to bring down an atrophic, white, scarred, fibrous testicle at puberty. Dr. Hinman, as well as Drs. Robinson and Engle, has placed the indications for orchiopexy in the absence of hernia on a scientific physiologic and histologic basis. We are in complete accord with the recommendation that the undescended testicle associated with inguinal hernia should be repaired early in life.

Drs. Robinson and Engle demonstrated last year, by repeat biopsy technic similar to that used by Dr. Hinman, that progressive spermatogenic damage supervened after the age of five years. They established indisputably the value of surgical placement into the scrotum of abdominal or inguinal testes early in life, whether the condition was unilateral or bilateral.

Variations in the method of surgical treatment need not be discussed at length, except to say that the Torek two-stage procedure has proved practical and effective for many years. Relatively late modifications pertain essentially to minor changes in technic. Recently Dr. Morton Mayer of Los Angeles suggested that the gubernaculum be carefully perserved in the dissection of the abdominal or inguinal testis, particularly when sufficient length of the spermatic cord could not be achieved, because he found that the cord could be transected with preservation of the viability of

the testicle through the blood supply from the inferior epigastric artery which coursed through the gubernaculum. Thus the testicle could be swung down to a scrotal position, using the gubernaculum as a fulcrum in preference to primary sacrifice of the testis by orchiectomy.

Dr. Hinman's paper provides effective corroboration of the thermophysiologic indications for orchiopexy, certainly before the age of six years and preferably even earlier. The psychologic and endocrine advantages to the patient of having scrotal testes before school age are obvious. Finally, since the undescended testis has a questionable tumor-forming potential greater than the normally located gland, another reason is offered for early surgical placement of the testicle into the scrotum.

Frederick L. Reichert (San Francisco, Calif.): Are you surgeons treating the patients by bringing the gonads down into the scrotum? You say that you administer large doses of gonadotrophic hormone for one week. The testis does not come down because it cannot, due to lack of sufficient hormone. You might administer something continuous to develop the pituitary.

If I hypophysectomize a puppy at one month of age, I find that the testes atrophy. We have more or less been trying to restore the adrenal gland, and we gave hypophysectomized puppies as well as their littermate controls large doses of vitamin C by injection. The pathologist would not get interested in the adrenals because he was concerned with the testes. These testes at the end of three months had normal sperm, whereas the littermate controls had none. That is another thing for consideration. If you really want to obtain sperm and have something worth while once you bring the testes down, help develop them with anterior pituitary hormone.

Wm. H. Snyder, Jr. (Hollywood, Calif.): Dr. Hinman has presented a very basic aspect of this problem and I commend him on a very good paper.

There is one problem that has not been settled as yet and I think he will approach it as the years go by. Robinson and Engle, and Hinman who has also been interested in the project, have determined that the size and development of the testis and of the spermatogenic tubules are delayed in the undescended testis. However, they have not as yet proved that even though you bring down the testes they will maintain their normal growth. Further proof of this will have to come about eighteen years later, when biopsies and fertility studies are done. That does not defeat our purpose (we must do it now on a theoretic basis) but it does not assure us that even with this early placement we are going to get the results we want.

Emile F. Holman (San Francisco, Calif.): How long have you observed the artificial testes that you place in the scrotum? How long have they remained without complication? I have heard of china eggs being put in the scrotum to replace the removed testes. We repair large hernias in old men in whom we remove the testis to insure permanent closure of the hernia. We could use such an artificial testis to good advantage.

Loren R. Chandler (San Francisco, Calif.): I would like to ask Dr. Hinman one question: What has been your experience with converting or convincing the pediatrician who usually takes care of these babies up to six, seven and eight years that operation is necessary and not long, repeated courses of hormonal therapy?

Robertson Ward (San Francisco, Calif.): I would like to ask Dr. Hinman if he decides what to do from a gross examination or from a microscopic section in fibrous disease of the undescended testis.

Frank Hinman, Jr. (closing): I am convinced that the testis should be brought down so that it can be watched in case seminoma develops. Seminoma is significantly more common in cryptorchidism. However, it can occur in either testis; it does not necessarily appear in the cryptorchid testis. Since some 60 per cent of patients with seminoma have a high follicle-stimulating hormone level, probably something is wrong with the whole organism.

Of course, orchiopexy does not conclude the surgeon's responsibility. Dr. Charney told me of a patient upon whom he performed orchiopexy. Seminoma later developed which metastasized to the inguinal nodes, where excision is very difficult.

Dr. Reichert raises the question of the effectiveness of pituitary hormones. It is certainly true that the course of chorionic gonadotrophin we give is a poor imitation of puberty. However, it does produce the characteristic change in the external genitalia, and clinically does effect the descent of the testis.

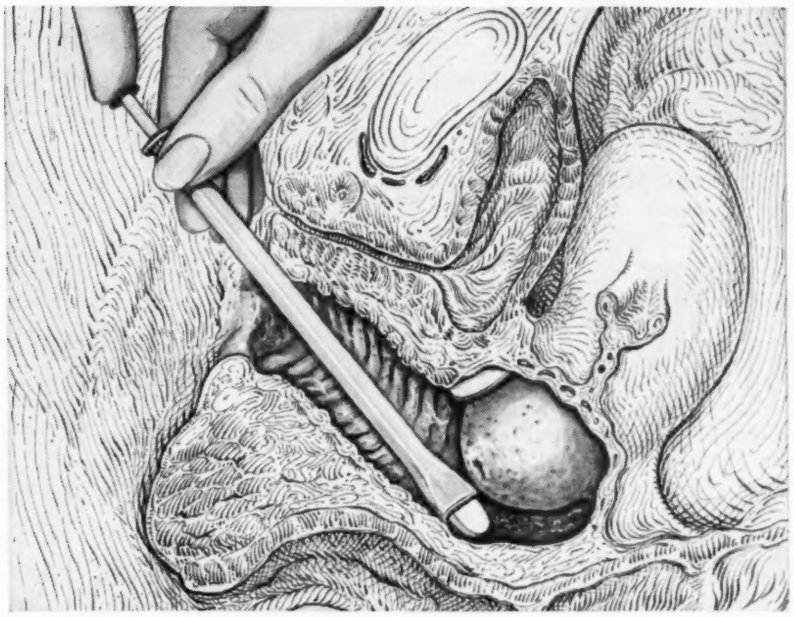
Dr. Snyder raises the really fundamental question which I think has become more pertinent in the last five years or so, i.e., will the testes function even if brought down. The only series so far that I know which gives even a partial answer is Dr. Hanson's from Scandinavia. He has shown that after unilateral orchiopexy he did not find any increase in fertility (he used the sperm count as a measure of fertility). With bilateral orchiopexy he did note improvement. However, his patients were not operated upon as early as we now advocate. It remains for someone who operates early to study these patients fifteen or twenty years from now.

Dr. Holman asks about the artificial testis. I think it is very worth while psychologically. I have used many more testicular prostheses in adults than I have in children. Because it is hard to

figure out in children how big they should be, I want to caution that we always seem to make testes larger than they should be; after they are in place they are often rather heavy. They are made of nylon. I keep one soaking in solution because often I do not know that I am going to remove the testis. If one is ready, you merely slip it into the scrotum, making a place for it with your finger, and it stays. Once in a while, if I do not stretch the scrotum sufficiently, it will be a little high and look a little uncomfortable, although no patients have complained. The longest I have followed up a patient is approximately five years; but if there is no infection, there is no difficulty.

Dr. Chandler asks how we are going to convert the pediatricians to earlier treatment. Well, I go around with a "message" all of the time. Conversion was one of the reasons for choosing such a topic for this meeting. The surgeon has to be convinced as well as the pediatrician. We must stress the progressive testicular damage and point out that even though we are not going to save all of the cryptorchid testes, we have our best chance when patients are young.

Dr. Ward asks how we are going to decide at the operating table if orchiectomy is necessary. Ordinarily orchiectomy is avoided unless there is an anatomic impossibility of bringing the testis down to the scrotum. If the testis is obviously embryonic, lying high retroperitoneally, it might as well be excised. At the same time, if the testis is of reasonably normal appearance, an attempt is made to bring it down. It does have slight androgenic function even though it will not add to the patient's fertility.



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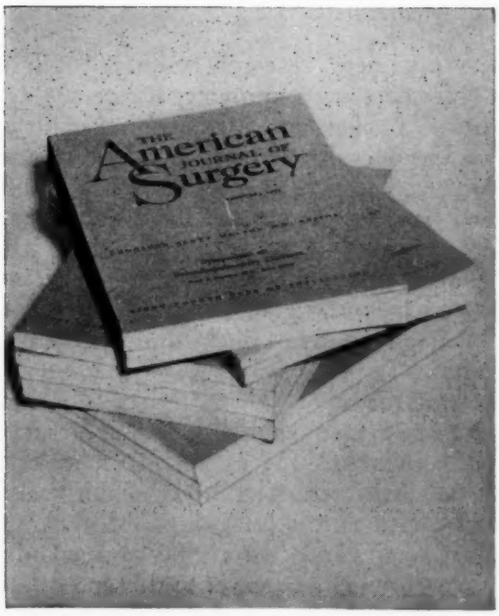

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NOTES AND QUOTES

A DRUG widely used in the treatment of tuberculosis may also serve as a preventive of the disease, if guinea pig experiments reported recently turn out to be applicable to human beings. The possibility of a dual role for isoniazid, the drug used with great success in treating tuberculosis, was suggested in a paper given at a medical session of the 51st Annual Meeting of the National Tuberculosis Association and the 50th Anniversary Meeting of its medical section, the American Trudeau Society.

Experiments with isoniazid as a prophylactic were reported by Dr. Carroll E. Palmer of the tuberculosis program, Division of Special Services, U.S. Public Health Service, and Mrs. Shirley Ferebee, statistician with the Division.

Guinea pigs given water containing isoniazid, then infected with tuberculosis, were found by the investigators to resist disease even when the daily ration of the drug was very small. Furthermore, when the drug was discontinued ten weeks after infection, the protection still appeared effective. The results in guinea pigs, Dr. Palmer contended, were better than with BCG, the most widely used vaccine against tuberculosis.

A new portable plastic carrying case for suture tubes, designed to fit safely into a physician's carrying bag and ready for use at on-the-spot emergency operations has been announced by Ethicon, Inc. The new clear plastic case which is 3⅛ inches wide, 5/6 inch deep and 4¼ inches high weighs just 4 ounces and is able to hold six tubes. It is constructed so that the inside walls are grooved to keep the tubes erect and separated from each other to avoid breakage. A drawn metal friction-fit cap is lined with a rubber strip to keep the tubes from rattling, as well as to compensate for the difference in suture tube lengths.

A FULL time eight-week comprehensive course in industrial medicine for physicians will be given in the Post-Graduate Medical School of New York University-Bellevue Medical Center starting September 26, 1955, as announced by Dr. Norton Nelson, Director of the Institute of Industrial Medicine.

Among the subjects being offered to physicians are: organization, administration and economics of an industrial medical department; the practice of preventive and constructive medicine in industry; the clinical aspects of occupational diseases; industrial injuries and the elements of safety programs; toxicology and industrial hygiene for the physician. Opportunities will be provided for attendance at medical, surgical and clinical pathologic conferences during the course. Applications should be sent to the Dean, N. Y. U. Post-Graduate Medical School, New York 16, N. Y. Tuition $250.

A thirty-four year old scientist has been presented the Endocrine Society Ciba Award for outstanding research in endocrinology, the study of body-regulating glands. Dr. Jack Gross, Associate Professor of Anatomy at State University of New York, Brooklyn, was honored at the annual meeting of the society for his discovery and study of a body chemical known as Compound No. 1, or tri-iodothyronine. The substance has proved a potent weapon in the treatment of underactive thyroid glands.

The Ciba Award, sponsored by Ciba Pharmaceutical Products, Inc., is made annually to a young scientist who, in the opinion of the Endocrine Society, has made a notable contribution in the field of endocrinology. This year's winner was selected by a panel of leading scientists headed by Dr. C. N. H. Long of Yale University School of Medicine.

TWENTY-ONE new research grants totaling nearly $218,000 were announced recently by Timothy V. Hartnett, Chairman of the Tobacco Industry Research Committee. These grants bring to over $490,000 the amount approved by the Committee for independent research projects into tobacco use and health, and for medical student fellowships. The Committee has set up a $1,000,000 fund from which grants are made, and has pledged more support as required.

"The need for expanded research of this kind becomes more evident each day," Mr. Hartnett said. "The cause of lung cancer is not known despite the ever-increasing research into this ailment. Scientific proof is lacking that smoking or any other single factor is the cause. Only science can provide the answer to this and other public health problems. It is our goal to help science find the facts."

CONTINUED ON PAGE 38

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NOTES AND QUOTES

Previous research grants announced by the Committee were for over $164,000 in January and for more than $82,000 in November. The Committee also has approved $26,500 for fellowships in 1955 designed to stimulate an interest in research in basic and experimental sciences among medical school students.

Research grants are made by the Committee at the recommendation of the Scientific Advisory Board, a group of nine noted doctors and scientists, which formulates and directs the Committee's research program and policy. Chairman of the Board and Scientific Director of the Committee is Dr. Clarence Cook Little, Director of the Roscoe B. Jackson Memorial Laboratory at Bar Harbor, Maine.

Dr. Little said eleven of the grants are for a cross-country co-ordinated lung study already under way. This project entails the collection of medical information at various hospitals in different parts of the country. The research is being co-ordinated by a member of the Scientific Advisory Board, Dr. Stanley P. Reimann, Director of the Institute for Cancer Research and the Lankenau Hospital Research Institute, Philadelphia. The statistical study of the collected data will be under the guidance of another Board member, Dr. Edwin B. Wilson, Professor Emeritus of vital statistics, Harvard University.

Duke University has just been granted $41,847 in American Cancer Society funds for support of research by five Duke scientists during 1955 to 1956. In all, $53,513 has been allocated in North Carolina, including $11,666 for grants at Wake Forest College and the University of North Carolina, in addition to the $41,847 at Duke. The $53,513 total is the largest ever granted by A.C.S. in the state since the program was started ten years ago. The over-all grant-in-aid program effective July 1st will provide $2,270,210 to finance 305 research projects at 120 institutions in thirty-six states and the District of Columbia.

A SCHOLARSHIP for undergraduates preparing for medical careers has been established at Hamilton College, in Clinton, New York, with a gift of $18,000 by the Lillia Babbitt Hyde Foundation of New York City. College President Robert W. McEwen, in announcing the gift, said that the scholarship is being offered in recognition of the special needs of undergraduate medical students for financial assistance.

The foundation has long been interested in medical research; the college has an excellent record for preparing men for careers in medicine and other fields of science.

This liberal arts college, which limits its enrollment to 600 men, was established in 1793 as the Hamilton-Oneida Academy by Samuel Kirkland, a missionary to the Oneida Indians. It was chartered as a college in 1812. Each year it sends over half of its seniors into graduate schools, with medicine, law and business the most popular fields.

At the annual meeting of the Pittsburgh Surgical Society in May, the Pittsburgh Surgical Society Award, in the form of a plaque, was presented for the first time. The recipient was Dr. Charles William Mayo, Professor of Surgery, Mayo Foundation, Rochester, Minnesota. After receiving the award, which will be presented annually in the future, Dr. Mayo addressed the members of the Surgical Society and their guests on carcinoma of the colon and rectum, with special reference to precursory lesions.

"THE alcoholic patient is now considered a sick person rather than a culprit, and the medical profession has finally conceded that the treatment of alcoholic persons is one of its responsibilities. Alcoholism is looked upon as a disease, or more particularly, as a symptom of various underlying pathologic changes." So declares Dr. Martha Brunner-Orne, in her article, "Treatment and Rehabilitation of Alcohol Addicts in a General Hospital Setting," which appeared in the June, 1955 issue of *Journal of the American Medical Women's Association.* Judging from a pilot study of over two hundred patients, carried out by the New England Hospital from March 1951 to June 1952, a short-term hospitalization makes it possible not only to treat the alcoholism but also to initiate rehabilitation . . . based on the threefold approach: physical, emotional and environmental . . . when the patient is most receptive.

CONTINUED ON PAGE 40

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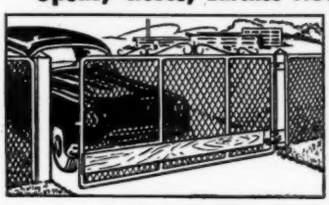

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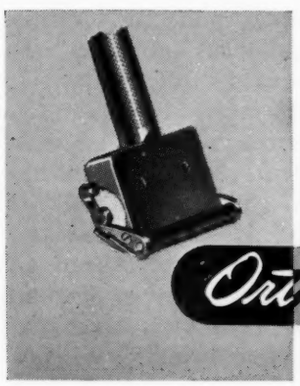

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"Once a person resorts regularly to intemperate use (of alcohol), so-called secondary symptoms develop and new complications are added to the underlying conflict situation . . . we try to break this vicious circle by relieving the secondary symptoms first. This can be done most efficiently during a short period of hospitalization when the patient is treated with sympathy and understanding in place of the contempt and punishment he has had to face in the past. In this hospital atmosphere, he more quickly regains his self-respect and confidence. He becomes amenable to suggestion and ready to accept an extended rehabilitation program.

"It is difficult to persuade an alcoholic when sober to seek help and treatment. However, during the acute stage of intoxication and during the period of hang-over, he is in need of medical attention and is then usually willing to submit to hospitalization. . . . While the patient is in the hospital, a rehabilitation program can be initiated.

"In each case, a physical examination is given. Drugs are administered to treat the acute stages, and therapy is focused first on a rapid and thorough physical build-up. One of the medications used recently is honey. "Honey contains a large amount of fructose which has been found to promote the metabolism of alcohol. The fructose-alcohol reaction has also a 'sobering up' and sedative effect on the patient, corresponding to the effect of vitamin B_6, and the combination of both has proved most helpful."

Short-term hospitalization is a positive contribution to the treatment of alcoholism, Dr. Brunner-Orne concludes, and it is hoped that other hospitals will be willing to accept alcoholics for treatment and for the initiation of a long-range treatment program.

John E. McKeen, president of Chas. Pfizer & Co., Inc., was honored by the government of France recently with the presentation of the Legion of Honor. The award was made in the course of ceremonies inaugurating Pfizer's newly built French plant. This was built in partnership with the French chemical firm of Clin-Byla, is geared to the production of the broad-spectrum antibiotics terramycin® and tetracyn, and will be able to produce a variety of other antibiotics, pharmaceuticals and chemicals. The installation is located at Massy, in the department of Seine et Oise, 8 miles south of Paris.

THE ORTHOPEDIC FRAME COMPANY of Kalamazoo, Michigan, recently announced the new Stryker Electro Surgical Unit consisting of six new lightweight attachments which snap conveniently on and off a single flexible cable and overhead motor. The tools are: (1) a bone drill, (2) screw driver, (3) rolo-dermatome, (4) rotary burring handpiece, (5) oscillating bone saw, and (6) a Seltzer rhinoplasty saw.

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The newly erected plant of Davis and Geck, Inc. in Danbury, Connecticut, was recently cited as "one of the ten most significant manufacturing plants constructed in the United States in 1954." Mr. C. P. Collins, Executive Vice President of Davis and Geck, accepted the award from Mr. C. W. Boyce, Associate Editor of Factory Management and Maintenance Magazine, sponsor of the nationwide competition.

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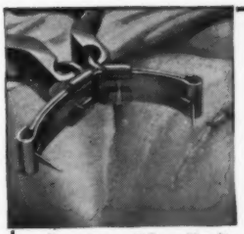

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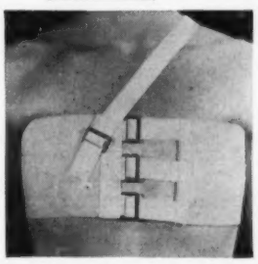

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